2 BIOLOGICAL BASES OF COGNITIVE DEVELOPMENT

IN THIS CHAPTER

Half of the first term had expired, and Tyler's first-grade teacher requested a conference with his parents. The teacher said that Tyler was a bright, creative, and likeable boy, but he was having some problems concentrating and staying on task. He would often blurt out answers to questions or say things that were not on topic, sometimes about one of his favorite TV shows. He was easily distracted and sometimes acted impulsively with the other children. After discussing some things his parents could do at home to help Tyler stay on task and suggesting that he likely did not have attention deficit hyperactivity disorder, the teacher asked the parents in what month Tyler was born. Tyler's father's first thought was, "*Uh oh, we've got a teacher who believes in astrology. What's next, tarot cards?*" But when they answered July, the teacher said, "I thought as much. He's a summer child, one of the youngest children in class, and a boy. His brain isn't as mature as most of the other children's. He'll catch up. He's a bright boy."

This experienced first-grade teacher may not have known that it was the slow-developing frontal cortex of Tyler's brain that was primarily responsible for his control problems or that psychologists refer to processing that involves staying on task, resisting interference, and planning as *executive function*. But this teacher had seen enough to know that "summer children," especially boys, were apt to get off to a slow start in first grade.

As I mentioned in Chapter 1, developmental psychology has become increasingly biological over the past few decades. For some time now, I've been including lectures on the biological

basis of cognitive development in my undergraduate class, discussing brain development and the evolution of mental abilities. This has not always been so. For much of the 20th century, social and behavioral scientists interested in cognition gave only lip service, at best, to biology. The mind might emanate from the brain, but understanding the brain was not seen as a prerequisite to understanding the mind. In fact, there existed in the social and behavioral sciences what can be called *biophobia* and an implicit belief that acknowledging biology was akin to rejecting the influence of environment or culture on behavior, something at odds with the central theme of the social sciences (see Tooby & Cosmides, 1992). The study of cognition was essentially isolated from the study of the brain.

Things have clearly changed. The field of cognitive science takes as a given the close connection between mind and brain. As philosopher John Searle (1992) stated, "Mental phenomena are caused by neurophysiological processes in the brain and are themselves features of the brain. . . . Mental events and processes are as much part of our biological natural history as digestion, mitosis, or enzyme secretion" (p. 1).

Looking at the biological basis of cognition and its development does not mean that one ignores the psychological level. Biology and psychology provide different levels of analysis. Much as psychology and anthropology present different pictures of human behavior (one at the level of the individual and the other at the level of the culture), so too do biology and psychology. Moreover, just as concepts in biology must be consistent with the known facts of chemistry, concepts in psychology must be consistent with the known facts of biology. Thus, proposing theories of the mind that are inconsistent with what we know about physiology or evolution cannot lead to a productive theory of cognition.

Psychology, however, cannot be reduced to biology. Knowing how nerve cells function will not tell us all we need to know about how we think. Developing a theory of the brain is important, of course, but it is not enough. Having a theory of the brain does not obviate having a theory of the mind. Cognitive psychology is not just something to do until the biologists get better at their trade. Developmental psychologists should not blindly accept everything that biologists propose, but they should be mindful of the biological causes of cognitive development and formulate theories and design experiments accordingly (Bjorklund, 2018).

In this chapter, I first describe evolutionary theory and how such Darwinian ideas can contribute to an understanding of the developing modern child. In the next section, I examine several developmental theories that take biology seriously, particularly the relation between genetic/biologic factors and environmental/experiential factors. I then provide a brief overview of brain development. In this chapter, as in later chapters, I comment on the relation between the brain and cognitive development. Although no one ever doubted that the brain was the seat of cognition, only relatively recently, with the emergence of the field of **developmental cognitive neuroscience**, have brain-cognition relations in development been taken seriously (see M. H. Johnson & de Haan, 2011). Developmental cognitive neuroscience takes data from a variety of sources—molecular biology, cell biology, artificial intelligence, and evolutionary theory, as well as conventional cognitive development—to create a picture of how the mind/brain develops. As will be made clear soon, contemporary biologically based theories of development do not hold that "biology is destiny" but, rather, deal with the classic nature/nurture

controversy by explaining how genes and environments interact to produce a particular pattern of development.

Because most research in cognitive development over the last century essentially ignored biological causation, most of what is covered in the rest of the book is at the psychological rather than the biological level. However, I firmly believe that we will develop an understanding of cognitive development only by taking biology seriously, and reference to biological factors is made throughout the remainder of the book.

EVOLUTION AND COGNITIVE DEVELOPMENT

What is the adaptive value of particular cognitive abilities? How might cognitive abilities have a different adaptive value at different times in development? In what contexts should certain cognitive abilities develop? How do some evolved human characteristics, such as bipedality or prolonged immaturity, affect the development of cognition? Developmentalists ask these questions related to evolution.

Evolutionary Theory

When biologists speak of **evolution**, they (usually) mean the process of change in gene frequencies within populations over many generations that, in time, produces new species. Modern evolutionary theory had its beginnings in the ideas of Charles Darwin, whose 1859 book *On the Origin of Species* represents one of the grandest ideas of science. The book made an immediate impact on the scientific community and is considered by many today to be one of the most important books ever written. The crux of the theory is that many more members of a species are born in each generation than will survive, and these members have different combinations of inherited traits (that is, there is substantial *variation* among members of a species). Conditions in the environment for a particular generation cause some members to survive and reproduce whereas others do not, a process that Darwin referred to as **natural selection**. The inherited traits of the survivors will be passed on to the next generation, whereas the traits of the nonsurvivors will not. Over the course of many generations, the predominant traits of a species will change by this mechanism. The major principle of Darwin's theory is *reproductive fitness*, which basically refers to the likelihood that an individual will become a parent and a grandparent.

Darwin's theory has gone through some substantial modifications during the last century or so, the most significant being the inclusion of modern genetic theory into formulations of evolution. Among scientists today, the *fact* of evolution is not questioned, although some lively debates center on the *mechanisms* of evolution (see S. J. Gould, 2002). Despite controversies, evolutionary theory is the backbone of modern biology, and because human cognition and behavior are rooted in biology, evolutionary theory should be the backbone of modern psychology.

One thing that evolutionary theory provides is a framework for interpreting all aspects of behavior and development. It does this, in part, by providing not only an explanation for *how* a particular mechanism came about (through natural selection) but also a possible explanation of *why* this mechanism evolved. In my early training, I was taught not to ask "why" questions.

Scientists, I was told, ask "how" questions—for example, "How do children come to appreciate that other people have perceptions and ideas different from their own?" rather than *why* do they develop this ability. Evolutionary theory provides answers to both the "how" and the "why" questions. The "how" is through natural selection over evolutionary time, in that children who could not learn to see the perspectives of another person did not grow up to have children of their own. Of course, this is not a sufficient answer to how this ability develops in individual children, but it does provide a mechanism for how it developed in the species. The "why" suggests that this ability was likely important for survival, or that it was *adaptive*. Children who could understand the perspective of another were able to anticipate other people's actions and act accordingly. Such *adaptationist* reasoning must be used cautiously, of course. Not all aspects of present-day life were necessarily adaptive for our ancient ancestors. Some aspects might have been neutral, some associated with other adaptive characteristics, and others just not sufficiently maladaptive to result in extinction. But having a theory that provides a framework for asking why a particular behavior or pattern of development is present can help us develop a better understanding of human nature and to ask better "how" questions.

It is important to understand that what might have been adaptive for our ancestors 10,000, 100,000, or 1 million years ago might not be adaptive for us today. Our preference for sweets and fat is a good case in point. Although these foods would have been rare and much valued sources of energy for our ancestors, they are easily available to people from postindustrial cultures today and are largely responsible for our high incidence of obesity and heart disease. Many cognitive mechanisms can be seen in a similar light. Alternatively, many of the technological problems we must solve as modern humans are only centuries old at most, and no specific mechanisms have evolved to solve them.

Evolutionary Developmental Psychology

Evolutionary theory is currently influencing cognitive development through the field of **evolutionary developmental psychology** (Bjorklund & Ellis, 2014; Bjorklund & Pellegrini, 2002; Hernández Blasi, 2020). Evolutionary psychologists have suggested that cognitive psychology is the missing link in explaining the evolution of human behavior. Leda Cosmides and John Tooby (1987) proposed that information-processing mechanisms evolved and that "these mechanisms in interaction with environmental input generate manifest behavior. The causal link between evolution and behavior is made through psychological mechanisms" (p. 277). According to Cosmides and Tooby, adaptive behavior is predicated on adaptive thought. Natural selection operates on the cognitive level— information-processing programs evolved to solve real-world problems. How do I tell friend from foe? When do I fight, and when do I flee?

From this viewpoint, it becomes fruitful to ask what kind of cognitive operations an organism must have "if it is to extract and process information about its environment in a way that will lead to adaptive behavior" (Cosmides & Tooby, 1987, p. 285). From an evolutionary perspective, we must ask what is the purpose of a behavior and the cognitive operations that underlie that behavior, and what problem was it evolved to solve.

Developmental Adaptations

What then evolved? Most evolutionary-minded psychologists argue that what evolved are *adaptations*, alterations in the structure or function of an organism that provided a survival or reproductive benefit. Adaptations can take the form of physical changes, such as an opposable thumb, greater fine-motor coordination, or a bigger brain. But not only do structures develop; so also do functions in terms of behavior, emotions, and cognition. These adaptations, however, do not function perfectly right out of the gate, but develop.

Some adaptations may serve not only to adapt infants and children to their immediate environment but also to prepare them for later environments. These are referred to as **deferred adaptations** (Bjorklund, 2015; Hernández Blasi & Bjorklund, 2003). For example, adaptations associated with establishing and maintaining social relations are important not only early in development in forming attachment with a primary caretaker, but also later in life in dealing with peers. Other types of adaptations may benefit the infant or child only during a specific time in development and then disappear when they are no longer needed. These are referred to as **ontogenetic adaptations**, and perhaps the clearest example of these would be the umbilical cord. The umbilical cord plays an essential role in keeping a fetus alive, transporting food, oxygen, and waste products to and from the fetus through the placenta. At birth, however, the umbilical cord's usefulness vanishes as the newborn's respiratory and digestive systems go through radical change.

Ontogenetic adaptations can be found in the psychological realm as well. One excellent candidate for an ontogenetic adaptation is **neonatal imitation**, in which newborns will (sometimes) match the facial expressions of an adult model, for example, sticking out their tongue after watching an adult stick out theirs (Meltzoff & Moore, 1977). Neonatal imitation is also observed in chimpanzees (Myowa-Yamakoshi et al., 2004) and monkeys (Ferrari et al., 2006) (see Photo 2.1). Neonatal imitation of facial gestures has been reported by a number of researchers (Heimann & Tjus, 2019; Nagy et al., 2020) and has been observed in infants with Down syndrome and newborns later diagnosed with autism spectrum disorder (Heimann & Holmer, 2021). However, the effect is somewhat elusive, and several experimenters have failed to replicate early imitation using procedures similar to those used by Meltzoff and Moore (Anisfeld et al., 2001; Oostenbroek et al., 2016). Melzoff and Moore (1977) originally proposed that neonatal imitation reflected true social learning, although this interpretation has been seriously questioned. For example, for the most-studied facial gesture of tongue protrusion, the majority of investigators who have examined infants of different ages report a peak in imitation sometime during the first 2 months, followed by a decline within weeks to chance values (Abravanel & Sigafoos, 1984; Jacobson, 1979). Facial expressions such as tongue protrusions are also elicited by flashing stimuli (Jones, 1996; Legerstee, 1991), music (Jones, 2006), or a looming black pen or small ball (Jacobson, 1979), causing Susan Jones (2009) to propose that neonatal imitation is young infants' response to interesting or arousing stimuli rather than reflecting true imitation. Other researchers have reported that infants tested at 1, 3, 6, and 9 weeks showed no selective copying of facial gestures, being just as likely, for example, to display tongue protrusion in response to observing a model opening her mouth as to sticking out her tongue (Oostenbroek et al., 2016; Redshaw et al., 2020).

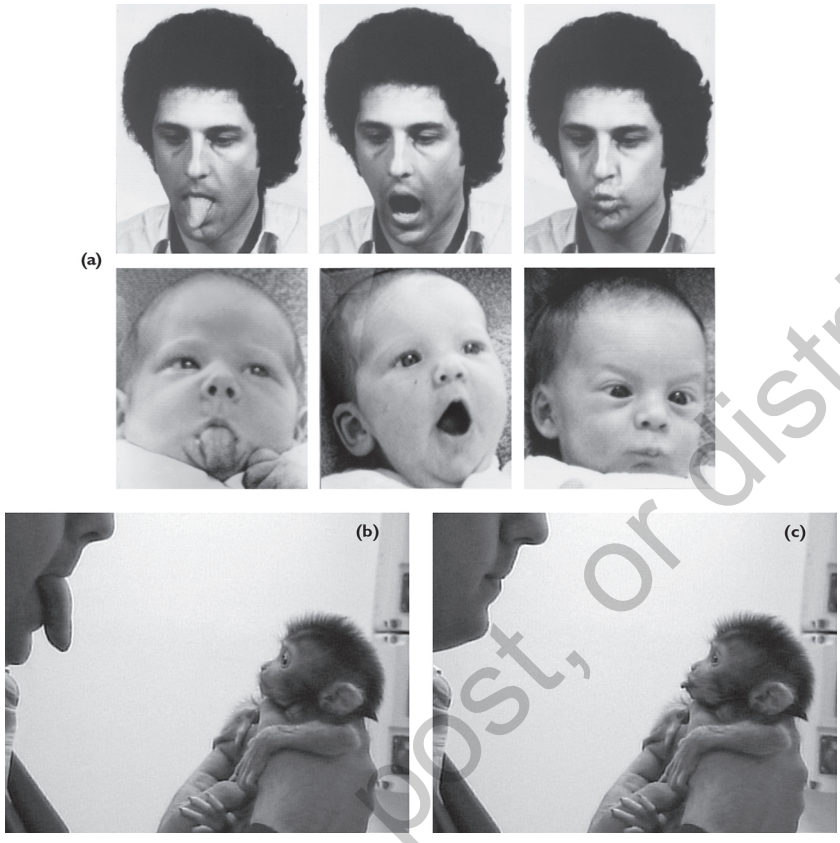


Photo 2.1 Neonatal Imitation in Humans (a) and Monkeys (b, c). Photographs of 2- to 3-Day-Old Infants Imitating (1) Tongue Protrusion, (2) Mouth Opening, and (3) Lip Protrusion as Demonstrated by an Adult Experimenter.

Sources: Gross, L. (2006). Evolution of neonatal imitation. *PLoS Biol4*, e311; Meltzoff, A. N., & Moore, M. K. (1977). Imitation of facial and manual gestures by human neonates. *Science*, *198*, 75–78.

If neonatal imitation is not a true form of social learning, what, if any, adaptive function might it have? The matching (or even nonmatching) of adult facial gestures by the infant might help maintain social interaction between the two, with these responses declining when infants are better able to intentionally direct their gaze and control their head and mouth movements in response to social stimulation, somewhere from the 2nd to 4th months of life (Bjorklund, 1987a, 2018; Byrne, 2005). In a similar vein, Maria Legerstee (1991) suggested that early imitation serves as a form of prelinguistic communication (see also Nagy et al., 2014). Mikael Heimann (1989) provided tentative support for these positions, documenting a relationship between imitation in newborns and mother-infant social interactions at 3 months of age (specifically, infants who showed high levels of neonatal imitation had more social interactions with their mothers 3 months later). Similar results of neonatal imitation predicting positive social behavior 1 month later has been reported for macaque monkeys (Paulkner et al., 2014).

The Emergence of Adaptations and Evolved Probabilistic Cognitive Mechanisms

It is also important to remember that adaptations, particularly deferred adaptations, *develop* and that the problems infants and children face are different from the problems adults face. Most mainstream evolutionary scientists assume that what evolved are domain-specific mechanisms designed by natural selection to deal with specific aspects of the physical or social environment, such as face recognition or the processing of certain types of social relationships. However, natural selection has also influenced the evolution of domain-general mechanisms (for example, executive function, ability to inhibit thoughts and actions), and a number of developmental psychologists believe that these should also be examined from an evolutionary perspective (Bjorklund & Kipp, 2002; Geary, 2005).

Implicit in the idea that there are domain-specific mechanisms is that there are *constraints* on learning (Spelke & Kinzler, 2007). Constraints imply restrictions, and restrictions are usually thought of as being bad. Human cognition is exceptional for its flexibility, not for its restrictiveness. But constraints, from this perspective, *enable* learning rather than hamper it. For example, several developmental scientists have proposed that infants are born with sets of perceptual or cognitive "primitives" related to processing information in a variety of domains, such as understanding the physical world (one object cannot go through another), processing numbers (1 + 1 = 2), or understanding the biological and social world. Concepts such as *starting-state nativism* (Gopnik & Meltzoff, 1997), *skeletal competencies* (Geary, 1995), *core knowledge* (Spelke & Kinzler, 2007), and *evolved probabilistic cognitive mechanisms* (Bjorklund et al., 2007) have been proposed to capture this phenomenon. From this perspective, humans are "prepared" by natural selection to process some information more readily than others (language, for example). But *prepared* is not *preformed* (Bjorklund, 2003). Instead, these constraints are the products of structured gene-× environment× development interactions that emerge in each generation and are influenced by prenatal as well as postnatal environments.

Let me expand on this idea by describing the concept of **evolved probabilistic cognitive mechanisms** (Bjorklund et al., 2007), defined as

> information-processing mechanisms that have evolved to solve recurrent problems faced by ancestral populations; however, they are expressed in a probabilistic fashion in each individual in a generation, based on the continuous and bidirectional interaction over time at all levels of organization, from the genetic through the cultural. These mechanisms are universal, in that they will develop in a species-typical manner when an individual experiences a species-typical environment over the course of ontogeny. (Bjorklund et al., 2007, p. 22)

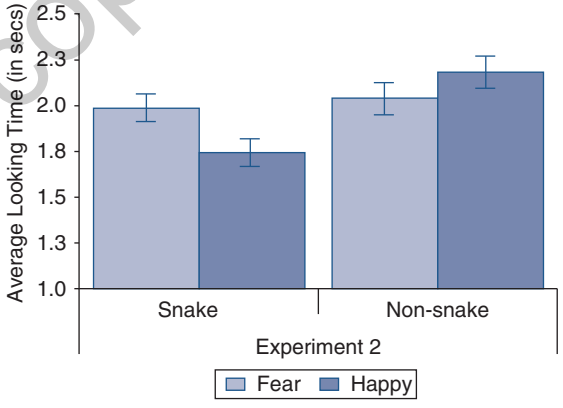
As an example of how evolved probabilistic cognitive mechanisms may work, consider the phenomenon of *prepared fear*. Monkeys raised in a laboratory show no fear of snakes. However, such monkeys are more likely to react fearfully after watching another monkey respond with fright to a snake than to a rabbit or a flower (Cook & Mineka, 1989), suggesting they are prepared to make fearful associations to snakes rather than having an innate fear of them (Rakison, 2022). Something similar seems to happen with human infants and children. For instance, Vanessa LoBue and Judy DeLoache reported that 3- and 5-year-old children (LoBue

& DeLoache, 2008, 2010) and 8- to 14-month-old infants (LoBue & DeLoache, 2010) more readily identified snakes or spiders among pictures of flowers or mushrooms than the reverse (something also found for adults; Öhman et al., 2001). Children even showed distinct patterns of evoked brain potentials to photographs of snakes compared to photographs of neutral stimuli (Hoehl & Pauen, 2017; Rakison, 2018). Yet children do not seem to have an innate fear of snakes but rather show a tendency to associate them with fearful responses. DeLoache and LoBue (2009) demonstrated this in studies in which 7- to 9-month-old infants and 14- to 16-month-old toddlers watched brief videos of snakes and other animals (for example, giraffes, rhinoceroses). The children initially showed no fear of the snakes. However, when the video clips were paired with either a happy or fearful voice, the toddlers looked longer at the snakes when they heard the fearful voice than when they heard the happy voice (see Figure 2.1). There was no difference in looking time to the two voices when they saw videos of other animals. This pattern of data suggests that, like monkeys, infants are not born with a fear of snakes. Rather they apparently possess perceptual biases to be attentive to certain classes of stimuli and to associate them with fearful voices, consistent with the idea of evolved probabilistic cognitive mechanisms (see Bjorklund, 2015).

Structure of the Mind

One way of thinking about how the mind is structured has been presented by David Geary (2005), who proposes that what evolved is a set of hierarchically organized, domain-specific

FIGURE 2.1 ■ Infants Looked Significantly Longer at the Snakes When Listening to a Frightened-Sounding Voice Than When Listening to a Happy Voice. Looking Times to the Other Animals Did Not Differ Significantly for the Happy and Frightened Voices.

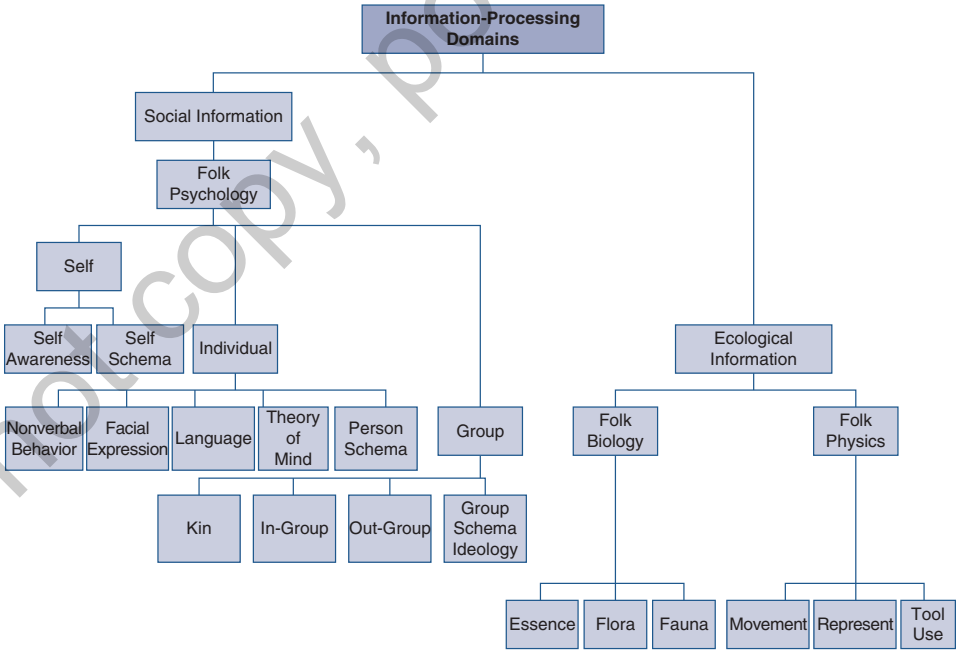


Source: DeLoache, J. S., & LoBue, V. (2009). The narrow fellow in the grass: Human infants associate snakes and fear. *Developmental Science*, *12*, 201–207 (Experiment 2).

modules that develop as children engage their physical and social worlds. Geary's model is shown in Figure 2.2. As can be seen, there are two overarching domains—social (folk psychology) and ecological—with each tapping into a limited pool of domain-general central executive resources and each consisting of more-specific domains (self, individual, and group for social; and biological and physical for ecological). Geary acknowledges that this list of domains is not complete (for example, there is no numerical domain listed here, which Geary believes exists), and one could argue about the organization of some of these domains. For example, should language be organized within the social domain, or is it best conceptualized as a separate domain? Nonetheless, Geary's organization reflects one that is consistent with the dominant perspective of evolutionary psychologists (Tooby & Cosmides, 2005), and it captures much of the developmental data.

Despite the belief that many evolutionarily influenced cognitive abilities are domain-specific in nature, one should not lose track of the fact that human cognition is amazingly flexible. This

FIGURE 2.2 ■ Geary Proposed That the Mind Is Hierarchically Organized Into Domains, With Lower-Level Modules, Designed to Process Less-Complex Information, Serving as Building Blocks for Higher-Level, More Complex, More Flexible Modules. Within the Social Domain of Folk Psychology, Domains Are Further Organized Into Those Dealing With (a) Self-Knowledge, (b) Individuals, and (c) Groups. Within the Ecological Domain, Geary Proposes Two Subdomains, One Dealing With the Biological World and the Other the Physical.



Source: Geary, D. C. (2005). *The origin of mind: Evolution of brain, cognition, and general intelligence*. American Psychological Association.

implies that what evolved in *Homo sapiens* are not highly specific approaches to problems but genes and cognitive mechanisms that are sensitive to different environments and that yield different outcomes (phenotypes) in different contexts that are (or would have been, in ancient environments) adaptive to local conditions. Such mechanisms become more specific and finely tuned during development, primarily as a result of experience. And humans, more than any other mammal, have time to gather the experience that will be necessary to function optimally as an adult. In fact, evolutionary developmental scientists have emphasized the importance of our species's extended childhood for cognitive development (Bjorklund & Pellegrini, 2002; Bogin, 2021). Humans spend a greater proportion of their life spans as juveniles than any other primate species. There are great dangers associated with delaying reproduction, however, so there must be some substantial benefits to survival for this prolonged period of immaturity to have been selected. Although there is no single answer to the question of why humans have such an extended juvenile period, one reason proposed by many evolutionary developmental scientists is that the long period of youth is necessary for children to master the technological and social complexities of human cultures (Bjorklund, 2021; Kaplan et al., 2000). This perspective argues that because of the variety of social and physical environments in which people live (both presently and in our evolutionary past), human cognition must be flexible, adapted not to a highly specific environment but to a broad range of potential environments, reflecting the diversity of social groups around the globe and throughout our species's history. To do this requires a long period of apprenticeship as well as a large brain capable of flexible learning and cognition.

Biologically Primary and Biologically Secondary Abilities

Another insight relevant to cognitive development and to education derived from evolutionary psychology is the idea that much of what we teach children in school is "unnatural," in that it involves tasks never encountered by our ancestors. For example, although our species has apparently been using language for tens of thousands of years, reading is a skill that goes back only a few thousand years, and only during the past century has a majority of people on the planet become literate. Geary (1995, 2005) refers to cognitive abilities that were selected over the course of evolution, such as language, as **biologically primary abilities**. Skills that build on these primary abilities but are principally cultural inventions, such as reading, are considered **biologically secondary abilities**. Biologically primary abilities are acquired universally, and children typically have high motivation to perform tasks involving them. Biologically secondary abilities, in contrast, are culturally determined, and tedious repetition and external pressure are often necessary for their mastery. It is little wonder that reading, a supposed "language art," and higher mathematics give many children substantial difficulty. (See Table 2.1 for a summary of the characteristics of biological primary and secondary abilities.)

It is important to emphasize here that an evolutionary account of development is *not* one of biological determinism. That is, although evolution works through changing frequencies of genes within the population, natural selection requires a dynamic interaction between organisms and their environments. Organisms choose environments, the very act of which modifies those environments. Environments in turn affect the organism by "selecting" some behaviors that "match" those environments over others. Because of this dynamic interaction between

TABLE 2.1 ■ Some Distinctions Between Biologically Primary and Biologically Secondary Abilities. Language Is a Good Example of a Biologically Primary Ability, Whereas Reading Is a Good Example of a Biologically Secondary Ability.

Biologically Primary Abilities
Have undergone selection pressure and evolved to deal with problems faced by our ancestors
Are acquired universally
Are acquired by children in all but the most deprived of environments
Children are intrinsically motivated to exercise biologically primary abilities and do so spontaneously
Most children attain "expert" level of proficiency
Biologically Secondary Abilities
Do not have an evolutionary history but are built on biologically primary abilities
Are culturally dependent, reflecting the cognitive skills that are important in a particular culture (such as reading in literate cultures)
Children are not intrinsically motivated to exercise these skills and must often be pressured by adults to acquire them
Tedious practice is sometimes necessary to master biologically secondary abilities

Source: Adapted from Geary, D. C. (1995). Reflections of evolution and culture in children's cognition: Implications for mathematical development and instruction. *American Psychologist*, *50*, 24–37.

organisms and environments, we must evaluate these interactions if we want to understand adaptation and cognitive development. Thus, this position rejects any simple notion of biological determinism (for example, "genes cause behavior") on cognitive development, intelligence, or the educability of children. An evolutionary perspective does *not* imply that adaptations represent fixed (that is, unchangeable) characteristics. In fact, evolutionary developmental scientists argue that *plasticity*, the ability to change as a result of experience, is an evolved characteristic of infants and children, resulting in children being sensitive to their early environments and adjusting their brains, cognitions, and behaviors accordingly (Bjorklund, 2021).

SECTION REVIEW

Developmental psychology has become increasingly concerned about biological causes of cognition.

Evolution and Cognitive Development

- Darwin's idea of variation and *natural selection* remains the cornerstone for theories of *evolution*.

- A central tenet of *evolutionary developmental psychology* is that both domain-specific and domain-general mechanisms have been modified over time as a result of natural selection.
- *Deferred adaptations* both adapt children to their immediate environments and serve to prepare them for future environments, whereas *ontogenetic adaptations* benefit the infant or child only during a specific time in development and then disappear when they are no longer needed.
- *Evolved probabilistic cognitive mechanisms* emerged to solve recurrent problems faced by ancestral populations; they are expressed in a probabilistic fashion in each individual in a generation, based on the continuous and bidirectional interaction over time at all levels of organization, from the genetic through the cultural.
- Geary proposed that the mind is hierarchically organized, with two overarching domains evolved to deal with social information (folk knowledge) and ecological information (folk biology and folk physics).
- *Biologically primary abilities* such as language have been selected for in evolution and are acquired universally by children in all but the most deprived environment; children are intrinsically motivated to execute them, and most children attain "expert" level of proficiency.
- *Biologically secondary abilities* such as reading do not have an evolutionary history but are built on biologically primary abilities; they are culturally dependent, children are not intrinsically motivated to execute them, and tedious practice is sometimes necessary for their mastery.

Ask Yourself . . .

1. What are the basic principles of an evolutionary approach to human development?
2. How is *prepared fear* an example of an evolved probabilistic cognitive mechanism? Can you think of another possible example?
3. How are biologically primary abilities different from biologically secondary abilities? Provide examples of each.

MODELS OF GENE-ENVIRONMENT INTERACTION

All self-respecting developmentalists believe that development is the result of an interaction between genetic/biologic factors and environmental/experiential factors. There is really no other alternative. Some theorists are more explicit about the nature of the interaction than others, however, and in this section I examine two approaches that look at gene-environment interactions and their consequences for development. Each approach posits that the child is an active agent in their own development, that development proceeds through the bidirectional effect of structure and function, and that the context in which development occurs is as important as the genes the individual inherits. The two approaches are the *developmental systems approach* (or *developmental contextualism*), as advocated by Gilbert Gottlieb (1992; Gottlieb et al., 2006) and others (see Witherington & Lickliter, 2016), and a theory based on research

in behavioral genetics, the *genotype → environment theory*, as presented by Sandra Scarr and Kathleen McCartney (1983; Scarr, 1992, 1993).

There are some important distinctions between the two approaches, centering mainly on the degree to which outside experience, influenced by one's genes, modifies the organism versus the degree to which a biological organism shapes its own development through epigenetic processes. Although debate between theorists in these two camps can be vigorous (see D. S. Moore, 2013; Scarr, 1993), the difference between the two approaches can be seen as a matter of degree. And the critical point for our purposes is that these two models take the transaction of biological and environmental factors seriously, making it clear that we need to give more than lip service to the interaction of the multiple factors that produce development.

Developmental Systems Approach

Concept of Epigenesis

The **developmental systems approach**, so called because it views development as occurring within a system of interacting levels, is centered on the concept of **epigenesis**: "Individual development is characterized by an increase in novelty and complexity of organization over time—the sequential emergence of new structural and functional properties and competencies—at all levels of analysis as a consequence of horizontal and vertical coactions among its parts, including organism-environment coactions" (Gottlieb et al., 2006, p. 211). (In biology, *epigenetics* also refers to the complex biochemical system that regulates gene expression, and I will discuss epigenetics briefly with respect to plasticity later in this chapter.) Epigenesis involves the action of genes, of course, but also the action of RNA, ribosomes, proteins, neurotransmitters, neurons, and so on, all in interaction with the environment, broadly defined. Central to the concept of epigenesis is the activity of the organism itself in influencing its own development; the organism's unique experiences can influence the activation of genes and lead to long-term alterations in the transcription of DNA (such as changes in the way that information contained in DNA about a protein sequence is translated by RNA during protein synthesis). Along similar lines, Gottlieb (1991a) stated that epigenesis reflects a bidirectional relationship between all levels of biological and experiential variables, such that genetic activity both influences and is influenced by structural maturation, which is bidirectionally related to function and activity. This relationship can be expressed as follows:

genetic activity (DNA ↔ RNA ↔ proteins) ↔ structural maturation ↔ function, activity

The point here is that functioning at any level influences functioning at adjacent levels. For example, genes clearly direct the production of proteins, which in turn determine the formation of structures, such as muscle or nerve cells. But activity of these and surrounding cells can turn on or off a particular gene, causing the cessation or commencement of genetic activity. Moreover, experience in the form of self-produced activity or stimulation from external sources can alter the development of sets of cells.

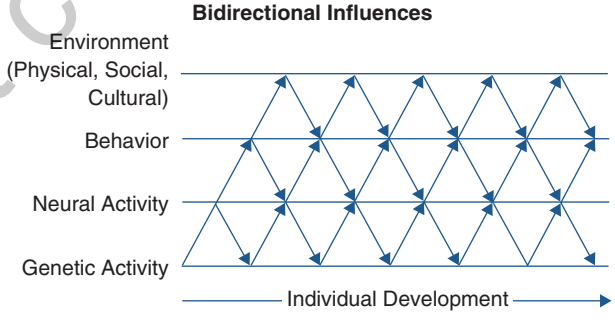
From this perspective, there are no simple genetic or experiential causes of behavior; all development is the product of epigenesis, with complex interactions occurring among multiple levels. Some compelling evidence for this claim comes from research involving twins. Identical

twins have identical DNA. Yet, as Mario Fraga and colleagues (2005) have shown, as twins age and undergo unique experiences, say differences in diet or tobacco use, they accumulate epigenetic differences (chemical differences affecting how genes are expressed). In their study, younger pairs of twins showed fewer markers of epigenetic differences than did older twins. These findings indicate that as even genetically identical individuals develop, their individual experiences can affect them at the cellular level. These cellular changes, in turn, can affect the expression of genes and behaviors, leading to further differences in experience. Such interactive effects lead to a cascade of changes across the life span, making it nearly impossible to distinguish environmental from genetic influences. This bidirectional approach to development is expressed in Figure 2.3. This figure suggests that we can never understand development merely by looking for genetic effects or for environmental effects; to understand development, we must look at the organism-context relationship. Mark Johnson (1998), in his review of the neural basis of cognitive development, made this point especially clear: "Since it has become evident that genes interact with their environment at all levels, including the molecular, there is no aspect of development that can be said to be strictly 'genetic,' that is, exclusively a product of information contained in the genes" (p. 4).

According to the developmental systems approach, new structures and functions emerge during development by means of self-organization through the bidirectional interactions of elements at various levels of organization (that is, genes, RNA, neurons, overt behavior, and so on). As Gottlieb (1991a) stated, "The cause of development—what makes development happen—is the relationship between the . . . components, not the components themselves. Genes in themselves cannot cause development any more than stimulation in itself can cause development" (pp. 7–8).

If the relations expressed in Figure 2.3 approximate reality, there should be substantial plasticity in development. Yet it is undeniable that development is constrained by one's genes.

FIGURE 2.3 ■ A Simplified Schematic of the Developmental Systems Approach, Showing a Hierarchy of Four Mutually Interacting Bidirectional Influences.

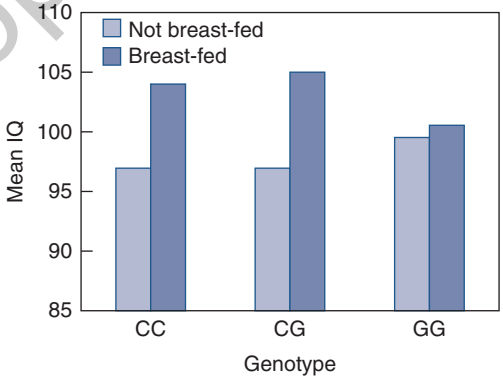


Source: Gottlieb, G. (1992). *Individual development and evolution: The genesis of novel behavior.* Oxford University Press. Used by permission of Oxford University Press, Inc.

Because our parents were humans, we develop in a way that a chimpanzee embryo can never develop, and vice versa. However, environments also constrain development. Genes will be expressed differently in different environments, yielding different patterns of development.

An example of how the effects of genes vary in different environments was provided in a study by Avshalom Caspi and his colleagues (2007), who examined the relationship among adult IQ, whether a person was breast-fed or bottle-fed, and specific versions of a gene associated with processing fatty acid. Previous research had documented a relationship between breast-feeding and later IQ, with children, adolescents, and adults who were breast-fed as babies having higher IQs than those who were bottle-fed (Mortensen et al., 2002). One explanation for this effect is that breast milk provides fatty acids (not found in cow's milk) that foster brain development early in life. Caspi and his colleagues (2007) identified a gene on chromosome 11 involved in processing fatty acids, as well as two variants of that gene. Recall from your basic biology class that we get one gene for a characteristic from our mother and one from our father and that the genes, or alleles, can vary somewhat (like having one gene for brown eyes and one for blue eyes). Children who had two combinations of the alleles (called CC and CG) *and* who were breast-fed as infants had significantly higher IQs (approximately 104) relative to children who had the same set of alleles but were not breast-fed (approximately 97). In contrast, children with a third version of the genes (called GG) showed no effect on IQ from being either breast-fed or bottle-fed (both groups had IQs of approximately 100; see Figure 2.4). Thus, the benefits of breast-feeding for subsequent IQ are influenced by a particular combination of alleles for a gene that influences how a person processes fatty acid. This study shows that even

FIGURE 2.4 ■ Relation Between Breast-Feeding and IQ for Children With Different Versions of a Gene for Processing Fatty Acids.



Source: Caspi, A., et al. (2007). Moderation of breastfeeding effects on the IQ by genetic variation in fatty acid metabolism. *Proceedings of the National Academy of Science, 104*, 18860–18865.

genes clearly associated with known specific biochemical and behavior outcomes (here, high IQ) are expressed differently in different environments (here, breast-fed versus bottle-fed).

If there is so much plasticity in development, why do almost all members of a species (human or otherwise) develop in a species-typical pattern? The answer is that a child (or a puppy or a duckling) inherits not only species-typical genes but also a species-typical environment. For example, ducks begin life in eggs, usually surrounded by other eggs, with their mother staying close by before they hatch. These ducks are able to hear and vocalize before hatching, and it turns out that these experiences contribute to an important aspect of posthatching behavior. Under normal conditions, when baby ducks, shortly after hatching, are put into a large container and hear the maternal call of two species of birds—their own and another—they invariably approach the call from their own species. They seem "instinctively" to know what their own species sounds like and to move toward that sound, something that makes good sense in the wild. However, when experimental procedures are performed so that the embryonic duck in the egg does not hear its mother or any of its siblings and its own vocal cords are temporarily prevented from functioning so that it can produce no sound itself, the duck fails after hatching to show the species-typical pattern of approaching the call of its own species (see Gottlieb, 1991b). In other words, prehatching experience, including hearing its own self-produced vocalizations, plays a major role in posthatching species-typical behavior. The reason that nearly all ducks approach the species-typical call after hatching is that nearly all ducks inherit not only the genetic disposition to make such a selection but also the species-typical environment that provides the necessary experiences for such a pattern to develop. Viewing development from this perspective provides a new meaning for the term *instinctive*. A behavior or function that is inborn in almost all members of the species might be instinctive, but if so, we must consider both the species-typical genes and the species-typical environment as factors contributing to that behavior.

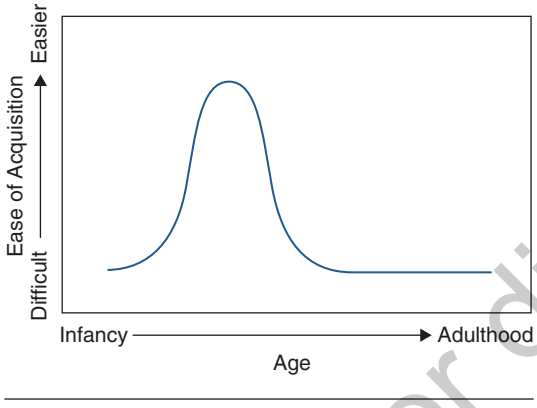
Results such as these indicate that behaviors (here, related to infant-mother attachment) found in almost all normal members of a species are influenced by often-subtle characteristics of the environment. Psychological mechanisms at the human level can be viewed similarly. Strong species-universal biases may exist for certain behaviors, but how any particular behavior or mechanism is expressed will depend on the experiences of the individual at certain times in development.

Developmental Timing

As any comedian will tell you, timing is everything. In the developmental systems approach, the timing of a particular event can influence substantially what effect that event will have on development.

Perhaps the concept most central to the issue of developmental timing is the **sensitive period**. The sensitive period (sometimes referred to as the **critical period**) for a specific skill or ability is the time in development (usually early in life) when it is most easily acquired. If a requisite experience occurs outside of this sensitive period (either too early or too late), the target skill will not be readily acquired—or possibly not acquired at all. Although the organism is most sensitive to a particular event at a particular time, similar or perhaps more intense experiences

FIGURE 2.5 ■ Some Cognitive Abilities, Such as Language, Might Be Most Easily Acquired During a Critical Period in (Usually Early) Development.



Source: © Cengage Learning

later in life can still have considerable influence on development. Figure 2.5 depicts the idea that a behavior is most easily acquired during a sensitive period.

Researchers have suggested that many aspects of human cognitive development can be described as involving sensitive periods, with language being perhaps the clearest example (Lenneberg, 1967; Newport, 1991). Both a first and a second language are acquired more easily when learned in early childhood. Although adolescents and adults can learn a second language, it is usually only with great difficulty, and they rarely attain the facility in that language as when it is learned during childhood. More will be said about a sensitive period for language acquisition in Chapter 9.

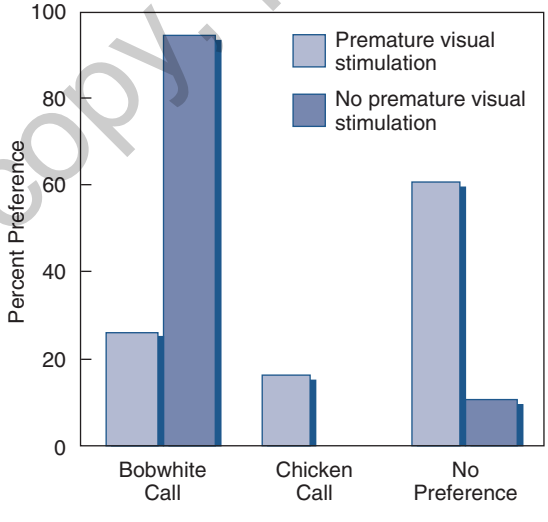
Examples of the significance of timing of perceptual experience come from research by Robert Lickliter (1990) involving auditory and visual stimulation of bobwhite quail. Like ducks, bobwhite quail approach the maternal call of their own species shortly after hatching. As demonstrated earlier by Gottlieb (1991b), this phenomenon has been attributed to auditory experiences the birds have before hatching. But, Lickliter reasoned, this is caused not just by the *presence* of auditory experiences before hatching but also by the *absence* of other sensory experiences, following an argument originally made by Gerald Turkewitz and Patricia Kenny (1982), who noted that the sensory systems in infants of many species function poorly at birth. Such inefficient functioning is actually adaptive, however, in that it protects the infant from sensory overload, permitting it to deal with small bits of simplified stimuli, which in turn makes it easier for the immature being to make sense of its world. Also, poor functioning in one sensory system (vision, for example) might permit an earlier developing sensory system (hearing, for example) to develop without undue competition for neural resources.

This is exactly the logic that Lickliter (1990) adopted for his study with bobwhite quail. In all vertebrates, hearing develops before vision. Lickliter argued that the slower development of vision in bobwhite quail allows the auditory system to develop without competition from the visual system. If so, what would happen if quail were given extra visual experience before hatching? One possibility is that it might hinder important aspects of auditory development, such as showing a preference for the maternal call.

Lickliter (1990) developed a procedure whereby he removed part of the eggshell and provided visual experience to bobwhite quail 2 to 3 days before hatching. Control quail had the end of the egg opened but received no visual experience. This ensured that the behavior of the experimental birds would be caused not by removing part of the eggshell per se but by the additional visual experience the chicks received. Lickliter then examined those birds with an auditory preference test in which the birds were placed in an oval container having speakers at opposite ends. From one speaker came the maternal call of a bobwhite quail, and from the other speaker came the maternal call of a chicken. The researchers observed which speaker, if either, the chicks approached.

The results of this experiment are shown in Figure 2.6. The control birds demonstrated the species-typical pattern when tested, with nearly all the birds showing a preference for the

FIGURE 2.6 ■ Percentage of Bobwhite Quail Chicks That Approached the Bobwhite Maternal Call, Approached the Chicken Maternal Call, or Showed No Preference as a Function of Whether They Received Premature Visual Stimulation.



Source: Adapted from Lickliter, R. (1990). Premature visual stimulation accelerates intersensory functioning in bobwhite quail neonates. *Developmental Psychobiology*, *23*, 15–27.

maternal call of their own species (that is, approaching the speaker from which the bobwhite quail call came). This was not the case for the birds that had the extra visual experience, however. A majority of these birds showed no preference or approached the speaker producing the maternal call of a chicken! I should note that these animals also displayed greater visual discrimination abilities. That is, the prehatching visual experience resulted in enhanced visual abilities, but at the expense of auditory abilities, which are important in the chick's development of attachment.

This and other studies (see Bjorklund, 1997) clearly demonstrate that the timing of perceptual experience is critically important and that earlier experience is not always better experience. This is worth remembering for human infants. Might their sensory limitations actually be adaptive, and might extra stimulation in one modality interfere with development in other modalities? Recall our discussion in Chapter 1 on the adaptive nature of cognitive immaturity and the demonstration that providing an infant with too much stimulation or "learning tasks" too soon in development might have a negative effect.

Although almost no research has been conducted with human infants on the topic of sensory overstimulation, researchers have speculated that some of the deficits experienced by premature infants are caused by exposure to too much sensory information too soon. Neonatologist Heidelise Als (1995) suggested the early sensory stimulation that premature infants experience might adversely affect brain development by requiring these infants to process perceptual stimulation that they would not normally deal with for several more weeks (see also Lickliter, 2000). As in the research with quail chicks, "premature" stimulation might result in enhanced performance later in life in some domains but at the expense of functioning in others, which often leads to forms of learning disabilities. And in fact, Als notes that these deficits are often accompanied by accelerated development or enhanced abilities in other areas, such as mathematics. This idea is provocative, although still speculative. But it is consistent with the idea that the timing of developmentally sensitive periods in the brain is correlated with the species-typical timing of perceptual experiences. According to Bjorklund and his colleagues (2007),

> When animals receive stimulation from one modality earlier than "expected" (i.e., when neural development and sensory experiences are uncoupled), it interferes with this choreographed dance between gene-influenced neural maturation and perceptual experience. This change in the gene-environment relation (in this case, a change in *timing* of different perceptual experiences) causes a species-atypical pattern of development. (p. 13)

Genotype → Environment Theory

Related to the developmental systems approach are several theories that stem from the field of **behavioral genetics**, which studies genetic effects on behavior and complex psychological characteristics such as intelligence and personality (Plomin et al., 2012; Rutter, 2006). These theories have attracted much attention among mainstream developmentalists, in part because they use human behavioral outcomes such as personality or IQ scores as data rather than generalizing

results from ducks, rats, or bobwhite quail to humans. This is also a reason for the substantial controversy the approach has produced (see D. S. Moore, 2013).

Academic psychologists have long been reluctant to accept a strong influence of genetics on human behavior. The argument against a genetic influence on behavior goes something like this: If we are what our genes determine us to be, then there is little hope of modifying the human spirit or human behavior through environmental intervention. If genes affect not only blood type and eye color but also behavior, personality, and intelligence, then biology truly is destiny.

Yet biology rarely dictates anything in an absolute way. As illustrated by Figure 2.3, all genetic effects are moderated by environmental ones. Even the genes for eye color must be expressed in a developing embryo, which is exposed to uncountable environmental factors as a result of its own development. The fact that genes influence behavior does not mean that environment plays only an inconsequential role. To deny the significant role of genetics in behavior is to place one's head in the sand, but to proclaim that genetics determines our personalities, intellects, and behavior is to seriously misinterpret reality.

Genotype → Environment Effects

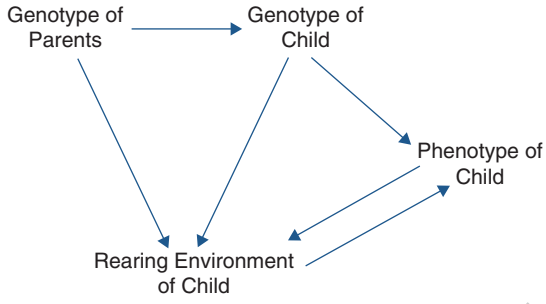
One of the most influential theories from behavioral genetics with respect to cognitive development remains Sandra Scarr and Kathleen McCartney's (1983) **genotype → environment theory**. Basically, Scarr and McCartney propose that one's genotype (one's actual genetic constitution) influences which environments one encounters and the type of experiences one has. Their basic contention is that *genes drive experience*. One's genetic makeup determines how one organizes one's world. Thus, environment does play a significant role in shaping intellect, but a person's inherited characteristics largely determine what those experiences are and how they are perceived.

Figure 2.7 presents a schematic of Scarr and McCartney's model of behavioral development. A child's phenotype (their observed characteristics) is influenced both by the child's genotype and by their rearing environment. The child's genotype is determined by the genotype of their parents. The parents' genotype also influences the environment; the parents' genetic characteristics affect the types of environments they feel most comfortable in. But in this model, the child's genotype also has an impact on the environment, which affects the child's development. Thus, characteristics of the child, as well as the rearing environment and genetic contributions of the parents, influence the course of development.

Scarr and McCartney posit three types of genotype → environment effects that vary in influence over time (see Table 2.2). They are *passive, evocative*, and *active*. Passive effects occur when genetically related parents provide the rearing environment of the child. When biological parents rear a child, the effects of genetics and environment cannot be separated because the people who provide the genetic constitution for a child also provide the environment. The influence of passive effects is proposed to decline with age.

Evocative effects occur when the child elicits responses from others that are influenced by his or her genotype. For example, an irritable child is responded to differently than a well-tempered child is, and the type of attention received by an infant who likes to cuddle is

FIGURE 2.7 ■ Scarr and McCartney's Model of Behavioral Development.



Source: Adapted from Scarr, S., & McCartney, K. (1983). How people make their own environments: A theory of genotype-environment effects. *Child Development*, *54*, 424–435. Copyright © 1983 The Society for Research in Child Development, Inc. Adapted with permission.

TABLE 2.2 ■ Three Types of Genotype → Environment Effects in Scarr and McCartney's Genotype → Environment Model.

Passive: Biological parents provide both genes and environment for children. Passive effects *decrease* with age.
Evocative: Temperamental characteristics of children evoke responses from others. Evocative effects remain *constant* with age.
Active: Children seek out environments consistent with their genotypes. Active effects *increase* with age.

different from that received by an infant who does not want to be held. During early childhood, an attentive and cooperative child receives more positive interactions from parents and teachers than an uncooperative, distractible child does. Evocative effects presumably remain constant throughout development.

Active effects occur when one's genotype influences the type of environments one chooses to experience. Individuals actively select an environment in which they feel comfortable. For example, children interested in competitive sports would probably seek out other like-minded children to play with who would be very different from the type of people sport-phobic children would seek. Accordingly, people with different genotypes choose to interact in different environments and, thus, have different experiences that influence their development. Active effects increase with age as children become increasingly independent of their parents and able to select their own environments.

How does Scarr and McCartney's model relate to cognitive development? For one thing, this model suggests that parents' environmental influence on children should be greatest during the early childhood years and decrease with age as active genotype → environment effects increase. Evidence for this position comes from an adoption study by Scarr and Richard

Weinberg (1978). They reported that the average correlations of the IQs of samples of adopted siblings (that is, genetically unrelated children living together) measured in early childhood ranged from 0.25 to 0.39. This indicates a moderate level of similarity between the IQs of biologically unrelated children growing up together, a reflection of an environmental influence on IQ. However, the correlation of the IQs for adopted siblings measured late in adolescence was 0! This means that knowing the IQ of one child would not help you predict, to any degree, the IQ of their adopted sibling. The predictive power is zero. These results reflect the fact that the longer these genetically unrelated siblings lived together, the less alike in IQ scores they became. Similar findings of reduced correlations of IQs with age have been reported for dizygotic (nonidentical) twins. Correlations of the IQs of dizygotic twins computed during the preschool years ranged from 0.60 to 0.75 but were reduced to 0.55 when measured later in childhood (Matheny et al., 1981). In fact, siblings in general become less alike in most respects the older they get (Sundet et al., 2008). Following Scarr and McCartney's (1983) model, passive genotype → environment effects, as reflected by the type of environments that parents provide for their children, decrease with age, and active genotype → environment effects increase. Why? Because as they get older, children are increasingly able to select environments that suit their particular needs, and such selection is determined primarily by one's genotype.

But then, do genes *cause* intelligence? Interestingly, Scarr and McCartney's theory ends up giving the environment a substantial role in directing development. Genotype causes a child to choose certain environments that are compatible with the child's genetic constitution, and the experiences in these environments shape the child's cognition (and other important psychological characteristics). From this perspective, one's genes serve to select "appropriate" environments, but experience is actually responsible for crafting the intellect. The heritability of intelligence is discussed in greater detail in Chapter 13.

Scarr and McCartney's model illustrates how genetic and environmental factors might interact to produce different patterns and levels of intelligence. Particularly attractive about this model is its consideration of developmental effects. Genetic and environmental effects are viewed not as constants but as dynamic factors that have different effects on intelligence at different points in time. This theory in effect postulates a transaction between developmental function and individual differences. As children become more autonomous with age, the influence of genetic and environmental factors on individual differences changes.

SECTION REVIEW

Differences in models of gene-environment interaction center mainly on the degree to which outside experience, influenced by one's genes, modifies the organism versus the degree to which a biological organism shapes its own development through epigenetic processes.

Developmental Systems Approach

- The *developmental systems approach* centers on the concept of *epigenesis*, a bidirectional relationship between all levels of biological and experiential variables such that genetic activity both influences and is influenced by structural maturation, which is bidirectionally related to function and activity.

- Organisms inherit not only a species-typical genome but also a species-typical environment, and species-typical experiences early in life can greatly influence the course of development.
- Many early perceptual and cognitive abilities are governed by *sensitive periods*, those times in development when certain skills or abilities are most easily acquired.

Genotype → Environment Theory

- Scarr and McCartney's *genotype → environment theory* is based chiefly on research in *behavioral genetics* and proposes that genes drive experience.
- Three kinds of genotype → environment effects are proposed: *passive*, which occur when biological parents rear the child; *evocative*, which occur when characteristics of the child elicit responses from others; and *active*, which occur when children select environments in which they choose to interact.
- Passive effects decrease in influence over time, whereas evocative effects remain constant, and active effects increase.
- Data supporting this theory show that parents' environmental influence on their children's intelligence is greatest during the early years and wanes as the children approach adolescence.

Ask Yourself . . .

1. How is the concept of epigenesis incorporated into modern cognitive developmental psychology?
2. What are some of the ways researchers have shown that the timing of perceptual experience can affect development?
3. What are the three types of genotype → environment effects?

DEVELOPMENT OF THE BRAIN

The human brain is perhaps the most marvelous thing in the universe. Unlike the brains of any other species, ours provides us with self-awareness and a behavioral flexibility that has allowed humans to create culture and to adapt to a limitless diversity of environments. Other animal brains are quite impressive, but only the human brain has led to language, mathematics, physics, and art.

Differences in thinking between humans and other mammals are, of course, directly related to differences in their brains. But the human brain does not have any special structures that other mammals don't have. The major differences between human brains and those of other mammals are in the greater amount of area that is devoted to the cerebral cortex and the extended period of postnatal growth.

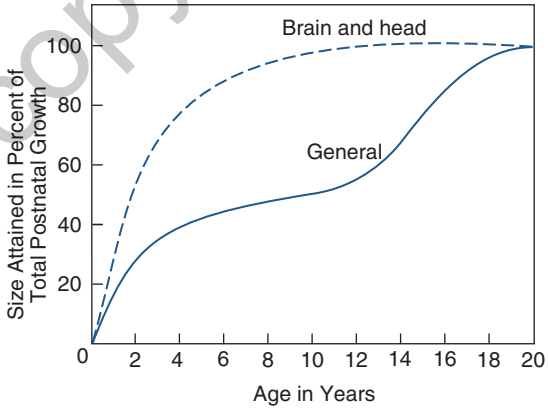
At birth, the human brain weighs about 360 grams—about 28% of its eventual adult weight. Compare this to overall body weight. At birth, infants weigh only about 5% of what they will weigh as adults. Stated another way, the brain accounts for about 10% of the overall body weight of a newborn but for only about 2% of the overall body weight of an adult. By 6 months,

the brain weighs 50% of what it will in adulthood; at 2 years, about 75%; at 5 years, 90%; and at 10 years, 95% (Lenroot & Giedd, 2007). In contrast, total body weight is about 20% of eventual adult weight at 2 years and only 50% at 10 years. So the brain, which grows rapidly before birth, continues its rapid development postnatally (Wilder & Semendeferi, 2022). The rapid postnatal growth of the brain and head relative to the body in general is depicted in Figure 2.8. From this perspective, babies are brainy creatures indeed. But from another point of view, the brain of a newborn is grossly underdeveloped. Although the brain works effectively enough to direct basic physiological functions (for example, breathing, wake/sleep cycles), it cannot control coordinated movement, and it cannot perform the mental operations so characteristic of our species. Despite its size, the infant brain is far from the organ it will become.

The human newborn's brain is large not only relative to its body size, but also relative to the brains of its nearest genetic relatives, the great apes. The average weight of a newborn chimpanzee's brain, for example, is about 135 grams compared to the 360 grams for a human newborn. However, human neonates' brains are actually a good deal less than their eventual adult weight, about 28%, than is typical for other primates, which is about 47% (DeSilva, 2022). This difference is due, in large part, to the fact that if a human newborn's brain followed the typical primate pattern, its skull would be too large to fit through the birth canal of a bipedal woman. This is referred to as the *obstetrical dilemma* (Washburn, 1960), and as a result, human infants are born "early" (with respect to proportion of eventual brain size, anyway), so that much brain development that would normally occur prenatally were humans to follow the typical primate schedule occurs postnatally in humans.

This phenomenon has been referred to by a number of terms, including *extrauterine spring* and *the fourth trimester* (Konner, 2010; Portmann, 1944/1990). As a consequence, human

FIGURE 2.8 ■ Growth Curves for the Brain and Head and the Body in General.



Source: Adapted from Scammon, R. E. (1930). The measurement of the body in childhood. In J. A. Harris, C. M. Jackson, D. G. Paterson, & R. E. Scammon (Eds.), *The measurement of man* (pp. 173–215). University of Minnesota Press.

infants' brains develop rapidly while being exposed to a broad range of physical and social stimulation that a typical primate would not receive until its brain was substantially more mature. These experiences, in turn, likely have a substantial influence on brain, cognitive, and social development and may be responsible, in part, for the extraordinary features of *Homo sapiens'* technical and social skills (Bjorklund, 2022; Portmann, 1944/1990; Trevathan & Rosenberg, 2016). As the German zoologist Adolf Portmann (1944/1990) wrote, "imagine the developing human spending the important maturation period of its first year in the dark, moist, uniform warmth of its mother's womb . . . It will gradually become clear that world-open behavior of the mature form is directly related to early contact with the richness of the world, an opportunity available only to humans" (p. 93).

The human brain, directly or indirectly, is responsible for controlling all aspects of behavior, from respiration and digestion to our most advanced forms of cognition. Our concern here is with the portion of the brain most associated with thought—the neocortex, or cerebral cortex. The neocortex is the most recent structure to appear in evolutionary time, associated primarily with mammals and having its greatest manifestation in primates and especially humans. Other areas of the brain—such as the limbic system, which is the seat of emotion—are also important and significantly influence human behavior, but the neocortex—particularly the *frontal lobes* (sometimes referred to as the prefrontal lobes) of the neocortex—provides the characteristics that we most associate with humanness. I discuss briefly certain aspects of the neocortex and its development later in this section.

Our knowledge of brain development and its relation to cognition has increased substantially during the past several decades, primarily because of new technologies that permit the imaging of brain activities (de Haan, 2015; Lenroot & Giedd, 2007). These **neuroimaging techniques** include, among others, *high-density event-related potentials*, which are a form of electroencephalography (EEG) that permit the detailed recording of brain activity when people solve cognitive tasks or are presented with specific stimuli; *positron-emission tomography* (PET) and *single-photon emission computed tomography* (SPECT), in which radioactive materials are injected into participants and changes in radioactivity are used to reflect glucose consumption in specific areas of the brain; and *functional magnetic resonance imaging* (fMRI), which is a noninvasive technique that measures blood flow to the brain while people perform cognitive tasks. A relatively new noninvasive technique that can be more easily used with infants and young children is *functional near-infrared spectroscopy* (fNIRS), which uses changes in near-infrared light between electrodes attached to the scalp to measure neuronal functioning. There are, of course, limits to such methods, but these and related new technologies promise that a new understanding of the relation between brain and cognitive development will soon be upon us.

I begin our discussion of the development of the brain by examining the basic building block of the brain—the neuron. I then examine how the brain gets "hooked up," the relation between brain development and behavior, and the role of experience in brain development and plasticity. This is not the last chapter in which brain development and the relation between children's brains and cognition is examined; many chapters devote space to this topic, for research in developmental cognitive neuroscience has expanded substantially over the past decade.

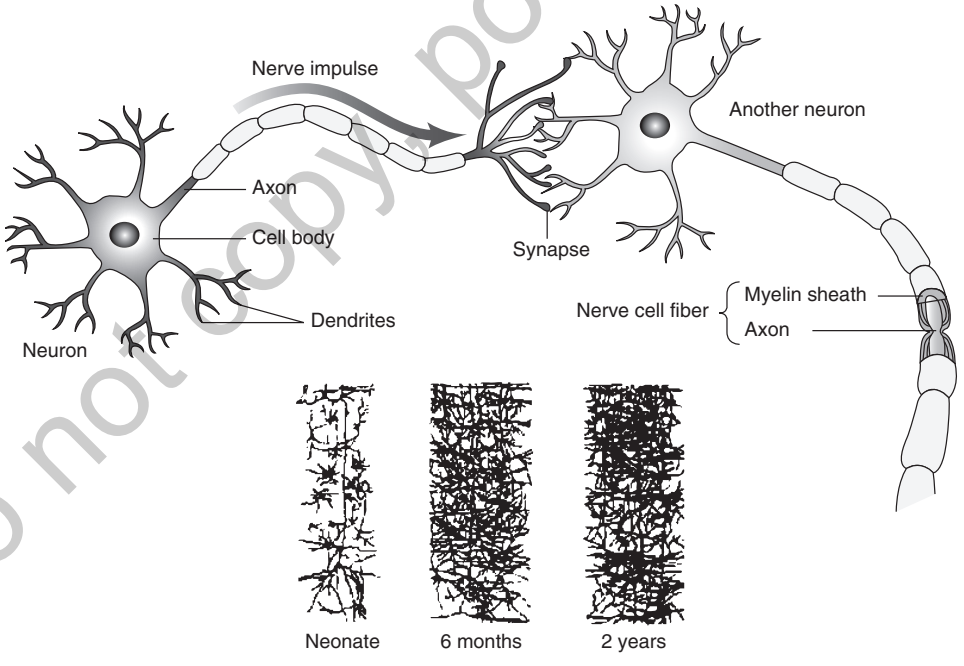
Neuronal Development

The brain, and the nervous system in general, is a communication system. Electrical and chemical signals are transmitted from one **neuron**, or specialized nerve cell, to another. A recent estimate puts the number of neurons in the mature human brain at 86 billion (Herculano-Houze, 2012). Unlike most other cells in the human body, neurons are not compressed together but are separated.

Figure 2.9 presents a drawing of a neuron. The main part of the neuron is the cell body, which contains the nucleus. Extending from the cell body are many projections, one of which is called the **axon**, a long fiber that carries messages away from the cell body to other cells. The other, more numerous fibers are called **dendrites**, which receive messages from other cells and transfer them to the cell body.

Dendrites do not actually come in physical contact with other dendrites (or with dendrite-like branches on axons, called *axon terminals*) when receiving messages. Rather, there are small spaces between dendrites, called **synapses**, through which messages are passed. The result is many billions of connections (synapses) among neurons. Electrical messages flowing down the axon of one cell cause the release of certain chemicals, called **neurotransmitters** (including *neuromodulators* that have an indirect effect on synaptic transmission), into the synapse. Neurotransmitters, which include dopamine, acetylcholine, and serotonin, move across

FIGURE 2.9 ■ Primary Structures of the Neuron.



Source: © Cengage Learning

the space between the cells and are "read" at the axon terminals of the adjacent cell, which convert the message back to an electrical signal and pass it on to its cell body. Conditions at the synapse (amount and type of neurotransmitters available) affect the transmission of the messages among neurons.

Fully developed axons are covered by sheaths of **myelin**, a fatty substance produced by supportive brain cells called *glial cells*. Like the plastic cover of an electric wire, myelin protects and insulates axons, speeding the rate at which nervous impulses can be sent and reducing interference from other neurons. Compared with unmyelinated fibers, myelinated nerve fibers fire more rapidly, have lower thresholds of sensitivity to stimulation, and have greater functional specificity, meaning that there is less "leakage" of electrical impulse so that only the target set of neurons is likely to get activated.

Myelination increases throughout childhood and adolescence, not being complete until sometime during the third decade of life or beyond. Myelination proceeds at different rates for different areas of the brain. For example, myelination begins prenatally for the sensory system, with most sensory structures being completely myelinated within the first year. This corresponds to the well-developed sensory abilities of human infants and the adultlike sensory capacities they possess long before they can speak. Myelination of the motor areas follows closely, with most of these brain structures being completely myelinated before the second year. Again, this corresponds to the development of motor abilities in young children, most of whom are walking before their second birthdays. The frontal cortex, the so-called thinking part of the brain, is the last to become fully myelinated, not being complete until early adulthood (Stiles et al., 2015).

When brains are stained with a chemical so that scientists can get a better look at their structure, myelinated areas appear white, whereas cell bodies and dendrites appear slightly pink or gray. This is a source of the terms *white matter*, reflecting mainly myelinated axons mostly beneath the surface of the brain, and *gray matter*, reflecting mostly cell bodies in both cortical and subcortical (that is, below the cortex) regions.

Proliferation, Migration, and Differentiation

Neurons go through at least three stages of development (Lenroot & Giedd, 2007; Stiles et al., 2015). The first stage is referred to as **proliferation**, or **neurogenesis**, which is the production of new neurons through the process of cell division by mitosis. During its peak, several hundred thousand neurons are generated *each minute* (C. A. Nelson et al., 2006). Proliferation occurs early in development, during the prenatal period. It was once believed that the 7th month after conception essentially marked the end of neuron production. However, subsequent research in both laboratory animals (E. Gould et al., 1999) and humans (P. S. Eriksson et al., 1998) indicated that new neurons are produced in adults at least in some areas of the brain, specifically the hippocampus, a structure that has been implicated in the formation of new memories. (New neurons are also generated in the olfactory bulb into adulthood.) In general, however, unlike other cells of the body, new neurons are typically not produced after birth. So with a handful of exceptions, people have all the neurons they will ever have at birth.

The second stage in neuronal development is **migration**. Once produced, the cells migrate, or move, to what will be their permanent position in the brain, where they collect with other cells to form the major parts of the brain. Not all cells migrate at the same time, but most cells have arrived at the final position in the brain by 7 months after conception (C. A. Nelson et al., 2006; Wilder & Semendeferi, 2022). Obviously, it is important that cells destined for a certain part of the brain be where they are supposed to be. Mistakes do occasionally happen, however, and faulty neural migration has been found to be associated with a variety of human disorders, including cerebral palsy, epilepsy, intellectual impairment, and learning disorders (Volpe, 2000).

The third stage in neuronal development is **differentiation** (or *cytodifferentiation*). Once at their final destination, neurons begin to grow in size, produce more and longer dendrites, and extend their axons farther and farther away from the cell body. Synapses are created during this stage. When an axon meets an appropriate dendrite from another neuron, a synapse is formed.

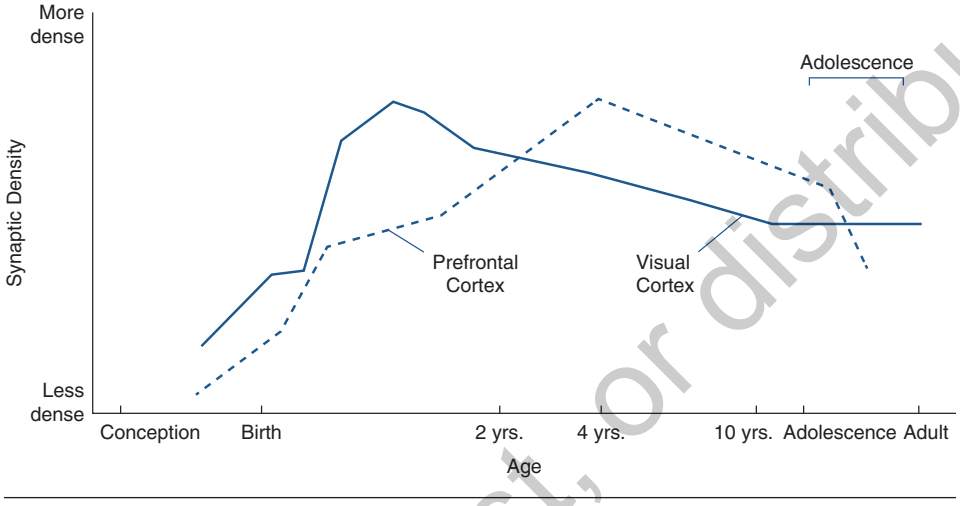
It is important to point out that differentiation does not stop at birth. In fact, most neuronal differentiation, particularly myelination (discussed earlier) and synaptogenesis (discussed next), takes place *after* birth (see Lenroot & Giedd, 2007; C. A. Nelson et al., 2006).

Synaptogenesis and Selective Cell Death

The process of synapse formation, or **synaptogenesis**, is rapid during the early years of life when the brain is first becoming organized. Synaptogenesis continues throughout life as the brain changes in response to new information, although the rate at which new synapses are formed is never as great as it is during those prenatal and early postnatal months when the brain is growing most rapidly. Synapse formation is perhaps more rapid in the months immediately following birth, but the peak of synapse formation varies for different parts of the brain. For example, a burst of synapse formation in the visual cortex begins at about 3 or 4 months of age and peaks between 4 and 12 months. At this time, the visual cortex has about 50% *more* synapses than there are in the adult brain. A similar pattern is found in the prefrontal cortex (the "thinking" part of the brain), but the peak number of synapses is not attained until about 24 months of age (P. R. Huttenlocher, 1994).

At this point, the infant brain has many more synapses and neurons than it needs, and a process of cell and synaptic pruning begins in earnest. (The pruning actually begins late during the prenatal period in a process known as **selective cell death, or apoptosis**.) Cell death and synaptic pruning occur at different rates for different parts of the brain. For example, the adult density of synapses for the visual cortex is attained from 2 to 4 years of age; in contrast, children continue to have more neurons and synapses in the prefrontal areas into their teen years than adults do (Stiles et al., 2015; see Figure 2.10). Thus, by their middle to late teens, adolescents have fewer, but stronger and more effective, neuronal connections than they did as children. Interestingly, the pattern of changes in cortical thickness (gray matter, mostly neurons) observed over childhood and adolescence varies with age and level of IQ, with this difference being especially pronounced in the frontal regions (Shaw et al., 2006). Researchers reported a negative correlation between cortical thickness and IQ in early childhood, such that children with higher IQs had thinner cortices than children with lower IQs. This pattern was reversed in late childhood and

FIGURE 2.10 ■ Age Differences in Synapse Production and Pruning in the Frontal and Visual Cortex. The Number of Synapses Show Sharp Increases Early in Development but Then Experience "Pruning," as the Brain Gets Sculpted to Its Eventual Adult Form. Note the Particularly Sharp Decline in Synapses in the Prefrontal Cortex During Adolescence.



Source: Huttenlocher, P. R., & Dabholkar, A. S. (1997). Regional differences in synaptogenesis in human cerebral cortex. *Journal of Comparative Neurology*, *387*, 167–178.

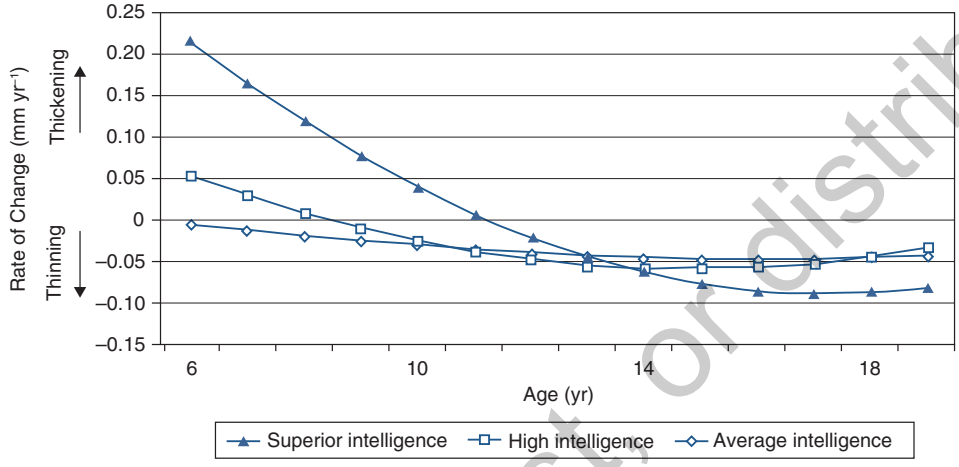
adolescence. Moreover, children with exceptionally high IQs showed an early acceleration of cortical growth followed by an accelerated thinning in early adolescence. This pattern of cortical thickness for children of average, high, and superior intelligence is illustrated in Figure 2.11.

Rises and Declines in Neural Development

The pattern just described for synaptogenesis is one of rapid development (that is, synapse creation) followed by a decline in the number of synapses (and neurons). Other aspects of brain development show a similar rise and decline over childhood. For example, the basic metabolism of the brain (the rate at which it uses energy) increases sharply after the first year of life and peaks at about 150% of the adult rate from ages 4 to 5 (Chugani et al., 1987). Evidence for this comes from studies using PET scans, which measure the amount of glucose uptake in the resting brain. After age 5 or so, the rate of glucose consumption slows down, reaching adult levels at about age 9. Thus, not only do infants and children have more neurons and synapses than adults, but their brains are also working harder (or at least using more calories) than those of adults.

In addition to changes in the actual structure of the neurons, developmental changes occur in the presence of various neurotransmitters—chemicals found in the synapses that promote the electrical/chemical communication between cells. Several of these neurotransmitters show increases followed by decreases over infancy and childhood, similar to the changes seen in synapses (M. H. Johnson, 1998).

FIGURE 2.11 ■ Rate of Change in Cortical Thickness for Children of Average, High, and Superior Intelligence. The Rate of Change for the Cluster of Cortical Points in the Right Superior and Medial Frontal Gyrus Showed a Significant Trajectory Difference Between Children of Average, High, and Superior Intelligence. Positive Values Indicate Increasing Cortical Thickness; Negative Values Indicate Cortical Thinning.



Source: Shaw, P., et al. (2006, March 30). Intellectual ability and cortical development in children and adolescents. *Nature*, *440*, 676–679 (p. 677).

What function might there be in this rise and fall in several aspects of brain development? One proposal is that the hypermetabolism seen during the preschool years might be necessary for the rapid learning that occurs during this time (Elman et al., 1996)—think of how children's language development proceeds from uttering only single words around 10 months to speaking in long paragraphs by age 3 or 4. Preschool children also have more neurons and synapses than older children and, I think it's fair to say, more to learn that is truly "new." These elevated levels of synapses and neurotransmitters also surely afford greater plasticity should brain damage occur. And although it may seem that slowing down the processes of synaptic pruning and cell death would afford children some advantages (more neurons and synapses can presumably do more learning), the failure to display such losses has been linked to intellectual impairment, schizophrenia, and other developmental disorders (Margolis et al., 1994).

A brief description of the major features of neuron development is presented in Table 2.3.

How Do Young Brains Get Hooked Up?

I have described briefly the process of synaptogenesis and related development of neurons, such as myelination. But how do brains actually get wired? That is, what mechanisms are responsible for building a brain that eventually will be able to recognize faces, solve arithmetic problems, talk, and read? Perhaps the position accepted implicitly by most brain scientists throughout

TABLE 2.3 ■ A Brief Description of Major Features in Neuronal Development.

Developmental Event	Timeline	Brief Description
Proliferation (neurogenesis)	First 20 weeks after conception	Neurons are born from neural stem cells. This peaks in the 3rd or 4th month of gestation.
Neural migration	6–24 weeks after conception	Neurons move, or migrate, to their "adult" location in the brain.
Differentiation/synaptogenesis	3rd trimester through adolescence	Neurons extent their dendrites and axonal terminals, forming synapses with other neurons.
Postnatal neurogenesis	Birth–adulthood	New neurons develop in some parts of the brain, including the dentate gyrus of the hippocampus and the olfactory bulb.
Myelination	3rd trimester to adulthood	Neurons become coated by a fatty tissue (myelin) that results in faster transmission of nervous signals and a reduction of interference.
Selective cell death (apoptosis)	3rd trimester to adulthood	Neurons die.
Synaptic pruning	Infancy–adulthood	The number of synapses per neuron is greatest between 4 and 8 months of life and decreases with age.

most of the 20th century was that the brain becomes specialized, and complex cognition thus arises, through intrinsic genetic and biochemical mechanisms. In other words, genes dictate the formation, migration, and differentiation of neurons, with experience serving only to "fine-tune" the brain. Few developmental neuroscientists believe this today, however, arguing instead that brain development involves an extended process that is greatly influenced by postnatal experience (Fox et al., 2010; C. A. Nelson et al., 2006).

It has become increasingly clear to those who study brain development that a reciprocal relationship exists between brain and behavioral development. To quote developmental neuroscientist Joan Stiles (2009), "Behavioral development is inextricably linked to brain development and vice versa. They are absolutely interdependent, exerting bidirectional influences that are essential for the normal development of the child" (p. 199). With respect to the survival of neurons and the process of synaptogenesis, William Greenough and his associates (Black et al., 1998; Greenough et al., 1987) proposed that specific experiences produce neural activity that in turn determines which of the excess synapses will survive (see also M. H. Johnson & de Haan, 2011). The nervous system of animals (including humans) has been prepared by natural selection to expect certain types of stimulation, such as a three-dimensional world consisting of

moving objects. I say "expected" in that these are the types of stimulation that virtually all of humans' ancestors experienced; that is, they are species-typical. Greenough and his colleagues referred to the processes whereby synapses are formed and maintained when an organism has species-typical experiences as **experience-expectant processes (or experience-expectant synaptogenesis)**; as a result, functions will develop for all members of a species, given a species-typical environment. Early experience of merely viewing a normal world, for example, is sufficient for the visual nervous system to develop properly. Those neurons and connections that receive the species-expected experience live and become organized with other activated neurons, and those that do not receive such activation die. Thus, although the infant comes into the world prepared and "prewired" both for certain experiences and to develop certain abilities, these abilities are substantially influenced by experience. What is hardwired seems to be a susceptibility to certain environmental experiences rather than the circuitry for detailed behaviors themselves.

Examples of experience-expectant processes can be seen in research that restricts the species-typical perceptual experiences of an animal. For instance, cats or rats reared in total darkness or in the absence of patterned light later have difficulty making simple visual discriminations. That is, because they were not exposed to visual stimulation early in life, when later provided with visual experience they act as if they cannot see, or at least do not see normally (Crabtree & Riesen, 1979). In humans, cataract patients who suddenly gain sight via surgery have difficulty making simple visual discriminations. For example, for several weeks after surgery, they can tell the difference between a square and a triangle only by counting the corners (Senden, 1960). Visual abilities for both animals and humans improve with time, but the longer the period of deprivation, the less reversible are the effects (Crabtree & Riesen, 1979; Timney et al., 1980).

The behavioral effects of sensory deprivation are reflected in changes at the neuronal level. For example, when a kitten's eyes first open, about half of the neurons in the visual cortex respond selectively to direction of movement or orientation of a stimulus (that is, firing only when an object in their visual field moves in a certain direction or is in a particular orientation, such as diagonal lines or straight lines). Usually, after several weeks of normal visual experience, all the cells in the visual cortex become sensitive to the orientation of a stimulus or to direction of movement. But when kittens are prevented from seeing any patterns (that is, when they experience only homogeneous light without any objects to see), the cells of the visual cortex make fewer connections with other cells and gradually lose their sensitivity to orientation. Experience (or lack of experience) changes the structure and organization of the young brain, even for something as basic as vision. As with behavior, recovery of normal neuronal structure and responsivity following exposure to pattern light occurs, although the amount or degree of recovery declines with longer periods of deprivation (Blakemore & Van Sluyters, 1975; Cynader et al., 1976).

One cannot do these kinds of experiments with children, of course, but research by Daphne Maurer and her colleagues with infants born having cataracts over their eyes, including some infants who had their cataracts removed shortly after birth, is informative (Le Grand et al., 2001; Maurer et al., 2007; see Maurer & Lewis, 2013, and Maurer, 2017, for reviews). Maurer and her colleagues reported that infants who had their cataracts removed and new lenses placed

in their eyes within several months of birth displayed a generally typical pattern of visual development. The longer the delay in removing the cataracts, however, the poorer vision was. Moreover, even for those infants who had their cataracts removed early and developed normal vision, some aspects of *face* processing were impaired (Le Grand et al., 2001). This finding suggests that there may be different sensitive periods for the brain areas associated with visual acuity and those associated with processing faces. It also points to the importance of identifying and correcting visual problems early to minimize their long-term effects.

Greenough and his colleagues (1987) proposed a second process of synapse development, which they called **experience-dependent processes (or experience-dependent synaptogenesis)**. In this case, connections among neurons are made that reflect the unique experiences of an individual rather than the experiences that all members of a species can expect to have. In both cases, the overproduction of neurons enables individuals to make connections (and, thus, store information) that reflect their particular environment. When certain experiences are not had—when the world does not cause certain neurons to be activated and synapses to join—the neurons die.

Bennett Bertenthal and Joseph Campos (1987) relate the ideas of Greenough and his colleagues to the old nature/nurture issue and the question of whether infants come into the world fully prepared by biology or as blank slates. Bertenthal and Campos write, "What determines the survival of synaptic connections is the principle of use: Those synapses activated by sensory or motor experience survive; the remainder are lost through disuse. For Greenough et al., then, experience does not create tracings on a blank tablet; rather experience erases some of them" (p. 560).

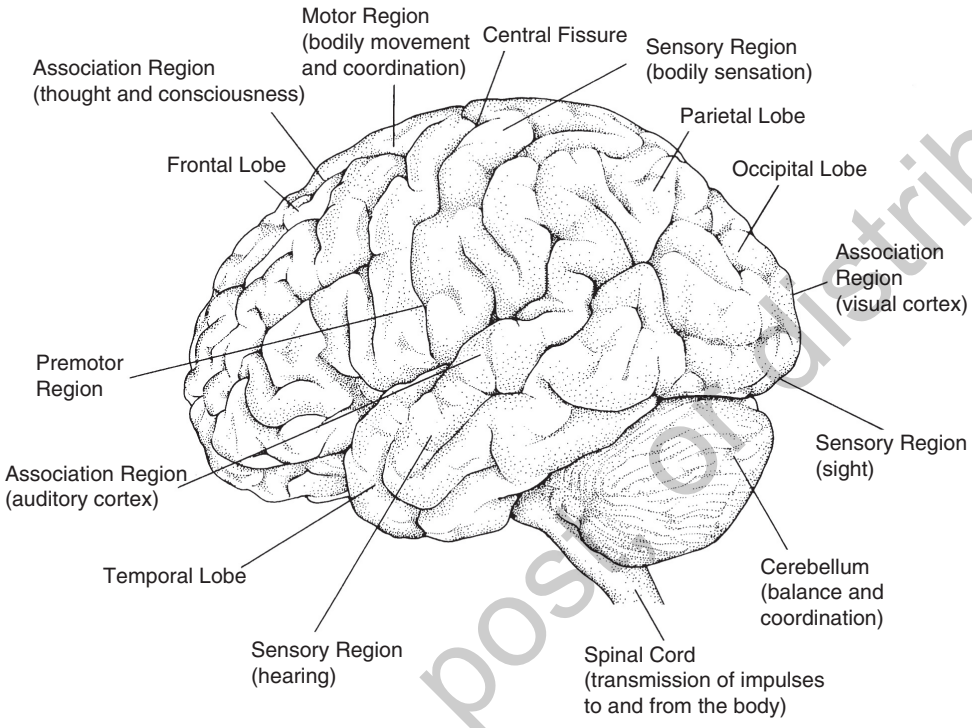
The message here is that early brain development is not exclusively under genetic control, consistent with the developmental systems approach discussed earlier. Certainly, genes influence what the basic structure of the brain will be. But experiences play an important role in shaping the precise circuitry of the brain. From electrical and chemical activities of the growing nerve cells before birth to the information obtained through the senses after birth, the brain becomes organized by information it receives and by its own activation as much as or more so than by the instructions emanating from the genes.

Development of the Neocortex

When most people think of the brain, they think of what's on the surface, a convoluted series of lobes. This is the **neocortex**, or **cerebral cortex**, which is a multilayered sheet of neurons, only 3 to 4 millimeters thick, that surrounds the rest of the brain. Figure 2.12 provides a lateral view of the brain; except for the cerebellum and the spinal cord, all of the structures shown are part of the neocortex. Of course, there is much more to the brain than the neocortex, but because the neocortex is the part of the brain primarily associated with thinking, it is the only major part of the brain that I discuss in this chapter.

The neocortex consists of two approximately equal halves, or hemispheres, connected by a thick mass of nerves called the **corpus callosum**. The neocortex can be further divided into regions. Some primary areas, such as the various sensory regions, receive information directly from the senses. Other primary areas, such as the motor regions, send instructions directly to

FIGURE 2.12 ■ A Lateral View of the Left Side of the Human Brain Showing the Major Structures. All but the Cerebellum and the Spinal Cord Are Part of the Neocortex.



Source: Bjorklund, D. F., & Bjorklund, B. R. (1992). *Looking at children: An introduction to child development* (p. 129). Wadsworth.

muscles. Secondary areas consist of regions that integrate information and have many connections with other areas of the brain. These are the association (or thought) regions, which are responsible for our more complex mental functioning.

Let me provide a research example of the connection between development of the prefrontal lobes of the neocortex and cognitive development. Development of the prefrontal lobes in humans is rapid between birth and about 2 years of age. As mentioned earlier, the prefrontal lobes are proposed to be involved in many acts of "higher" cognition, but one important function of the prefrontal lobes appears to be in the inhibition of responses (Fuster, 1989; see Carlson et al., 2013). For example, for some tasks, children must *not* execute a previously acquired response (that is, they must inhibit that response) so that they can make a new response. One such task is Piaget's A-not-B object permanence task. (See Chapter 4 for a more in-depth discussion of object permanence.) On this task, infants watch as a toy is hidden in one of two wells (A). The infants are then distracted for a delay period, after which they are allowed to retrieve the toy. Over trials, the hiding place is changed to Well B following a series of correct retrievals from

Well A. Piaget reported that infants younger than 12 months have great difficulty performing this task and typically look for the hidden object at the A location, where they were successful previously.

Adele Diamond (1985) tested 25 infants in the A-not-B task, beginning at about 7 months of age and continuing until 12 months. She reported that the delay between hiding and searching that was necessary to produce the A-not-B error increased with age at a rate of about 2 seconds per month. That is, 7.5-month-old infants would search for the hidden object at the erroneous A position following only a 2-second delay. By 12 months of age, infants made the error only if approximately 10 seconds transpired between the hiding of the object and the beginning of the search.

Although such research suggests that memory might be a factor in infants' performance, Diamond believed that the more important factor is infants' ability to *inhibit* prepotent responses in solving the A-not-B task. Substantial research with adults (Luna et al., 2001), older children (Barkley, 1997), and infants (M. A. Bell et al., 2007) points to the frontal lobes as the locus of inhibitory control, and Diamond (1991) proposed that during the first year, infants' prefrontal lobes develop gradually, which results in their becoming increasingly able to inhibit their behavioral responses. Despite "knowing" that the object was hidden at Location B, young infants cannot stop themselves from executing a response that has been correct in the immediate past. They have learned a response, and before they can learn a new one, they must inhibit the old one. This ability develops over the first year, with girls showing faster progress than boys, suggesting more rapid maturational development of the prefrontal cortex in girls through this age period (Diamond, 1985).

Support for Diamond's claim comes from a study showing significant relations between performance on the A-not-B task and scores on a task of inhibitory ability in 9-month-olds (Holmboe et al., 2008) and from neuroimaging studies that show connections between infants' performance on A-not-B tasks and frontal lobe activity (Baird et al., 2002; M. A. Bell & Fox, 1992). For example, Martha Bell and Nathan Fox (1992) recorded EEG activity from the frontal lobes of 7- to 12-month-old infants performing the A-not-B task. Consistent with Diamond's hypothesis, they reported systematic changes in EEG patterns as a function of age and length of delay.

Age-related changes in brain structure and function associated with changes in cognition and behavior are observed not only in infancy and early childhood but also later in life. It will likely not surprise you to learn that major changes in brain organization occur in adolescence, a time of substantial change in behavior and thinking. Although some, such as Piaget (see Chapter 5), have noted the advent of adultlike cognitive abilities during adolescence, a more common description of adolescent thought and behavior centers on a new self-centeredness, emotional instability, increases in risk-taking, and the seeking of novelty (see Spear, 2000). Not surprisingly, these behavioral changes are also associated with changes in the brain. For example, changes occur in the distribution of various neurotransmitters, with some decreasing substantially in both the frontal cortex and the limbic system, an area of the brain associated with emotion. Amount of gray matter in the adolescent brain actually decreases relative to childhood, while white matter increases (mainly due to increased myelination in the frontal

cortex), and different areas of the brain become increasingly connected (E. L. Dennis et al., 2013). Moreover, the amygdala and other structures in the limbic system reach adult levels before the prefrontal lobes (Mills et al., 2014). This produces what some researchers refer to as a mismatch in maturation (Giedd, 2015; Mills et al., 2014), which may be responsible in part for the sensation-seeking, risky behaviors and sometimes poor decision making often seen in adolescents.

These changes are likely adaptive, in that the emerging adult must seek independence from their parents, experiment with new environments, and establish a place in their social group (Sercombe, 2014). That there is a species-typical pattern of brain changes associated with such behavior should not be surprising. But what we have here is only correlation, and the correlation is far from perfect. Although many adolescents experience the storm and strife one might expect from a radical restructuring of the brain, others do not. There is a species-typical pattern of changes in cognition and behavior during adolescence, but there is also much variability. I am convinced that the brain mediates all such behavior; however, we must keep in mind that brain development is a dynamic process, influenced by both internal and external factors, rather than the simple consequence of the "unfolding" of a genetic blueprint.

The Brain's Plasticity

Plasticity, discussed in Chapter 1, refers to the ability to change. To what extent can new synapses be formed and different parts of the brain take over a function intended for another part of the nervous system? Put another way, plasticity refers to the potential outcomes that are possible for a single neuron, a bundle of neurons, or a larger brain structure. Given certain experiences at certain times in life, how might these cells become organized? Implicit in the theorizing of most developmental neuroscientists is the concept of plasticity.

Neuronal Plasticity

There is apparently little or no plasticity in the production of new neurons, at least in the cerebral cortex. As I noted earlier, new neurons in the hippocampus and olfactory bulb are generated throughout life, but evidence of neurogenesis in the cortex of humans and other mammals is scarce. With few exceptions, newborns come into the world with more neurons than they will ever need. And from birth on, there is a loss of neurons—a rapid loss during infancy and a gradual decline thereafter.

The picture is different for the formation of new synapses. Contemporary research indicates that new synaptic connections can be formed throughout life (Greenough et al., 1987; Guyer et al., 2018). What causes new synapses to form? The simple answer is experience. Perhaps the most convincing evidence of the effects of experience on brain structures comes from studies providing environmental stimulation for laboratory animals, mostly rats and mice. In studies dating back to 1949 (Hebb, 1949), researchers have raised groups of laboratory animals in environments constructed to be enriching or stimulating and then compared their brain development and learning ability with those raised in environments considered to be deprived (Turner & Greenough, 1985). Enriched environments usually included animals raised together in large cages that were filled with a variety of objects with which they could interact. Various platforms,

toys, and mazes filled some of the cages—not too dissimilar from the cages one might buy for the family gerbil.

These experiments have shown that rats and mice raised in enriched environments are superior at a wide range of complex tasks, such as maze learning. The differences in learning ability between enriched and nonenriched animals are of a general nature, with the most likely explanation for these effects being that "the groups differ in the amount of stored knowledge upon which they can draw in novel situations" (Greenough et al., 1987, p. 547). Concerning changes found in their brains, enriched animals have heavier and thicker neocortices, larger neurons with more dendrites, and, importantly, more synaptic connections. In one study, enriched rats had 20% to 25% more synapses per neuron in their visual cortices than did rats raised in individual cages (Turner & Greenough, 1985). And these effects are not limited to infant animals; the behavioral and brain benefits of living in a stimulating environment are found even when experienced by older animals (Greenough et al., 1986; C. A. Nelson et al., 2006).

Synaptic plasticity is greatest in infancy. With age and experience, neurons and synapses that were formed prenatally or in infancy die, and with their death, connections that could have formed are now impossible. Thus, experience serves not only to create new connections but also to make other ones impossible or less likely. But even though the plasticity to form new synapses decreases with age, it does not disappear; we retain substantial neural plasticity throughout life (Guyer et al., 2018). What does change is the degree to which experience can change the brain and the intensity of the experience needed to produce change.

The high degree of neuronal plasticity displayed by humans, especially in infancy, may seem contradictory to the idea of evolved domain-specific adaptations discussed earlier in this chapter. It is not. Neural plasticity is an evolved characteristic of *Homo sapiens*, and it extends later in life than in other primates. For instance, although chimpanzees and humans have similar genes associated with synapse formation in the cerebral cortex, the expression of these genes peaks earlier in chimpanzees (before 1 year) than in humans (about 5 years) (X. Liu et al., 2012). Perhaps of greater significance, gene expression associated with synapse formation is similar in human adult brains to that of juvenile chimpanzee brains (Bufill et al., 2011; Charrier et al., 2012; Somel et al., 2009), causing neuroscientist Enric Bufill and colleagues (2011) to propose that humans possess a form of **neuronal neoteny**. Neoteny refers to the retention of infantile or juvenile features, particularly of an ancestor, into later development. Bufill and colleagues (2011, p. 735) write that "human neurons belonging to particular association areas retain juvenile characteristic throughout adulthood, which suggests that a neuronal neoteny has occurred in *H. sapiens*, which allows the human brain to function, to a certain degree, like a juvenile brain during adult life . . . Neuronal neoteny contributes to increasing information storage and processing capacity throughout life, which is why it was selected during primate evolution and, to a much greater extent, during the evolution of the genus *Homo*."

I want to make it clear that losing plasticity should not be viewed completely negatively. As a result of genetic programming and experience, neurons become dedicated, or committed, to certain functions, effectively eliminating plasticity. This commitment affords greater efficiency of processing, permitting sets of neurons to specialize. For a species such as humans, who have long life spans and must deal with a large diversity of social circumstances, retaining some plasticity into adulthood is necessary. However, much about human life does not change

substantially over time and circumstances, and individuals are best served by a nervous system that early in life commits neurons to basic functions.

Recovery of Function From Brain Damage

Perhaps the best-known evidence for the plasticity of the nervous system comes from case studies of people who have experienced brain damage and exhibit deficits in physical or mental functioning. These studies document the process of readjustment these people go through and the differences their ages make to their readjustment.

Before proceeding, I must mention some problems that are inevitable when using brain-damage research to understand typical brain function. These include the facts that (a) brain damage can rarely be narrowed to one area; (b) brain damage frequently involves complications beyond simple lesions; (c) disorders following brain damage might not reveal how the brain functions normally; and (d) lesions in one area of the brain can lead to changes in other areas of the brain (Fuster, 1989). Nevertheless, despite these and other reservations, much can be learned about brain functioning from studying brain damage, especially when viewed in combination with other sources of data.

Research dating back to the middle 1800s has produced mixed results concerning age of brain damage and the likelihood of recovery, with some studies documenting greater recovery of function when brain damage occurs early versus later in life, and others showing just the opposite pattern (see V. Anderson et al., 2011). Two seemingly contradictory explanations have been offered to explain the relation between age of brain damage and subsequent recovery: *early plasticity* and *early vulnerability*.

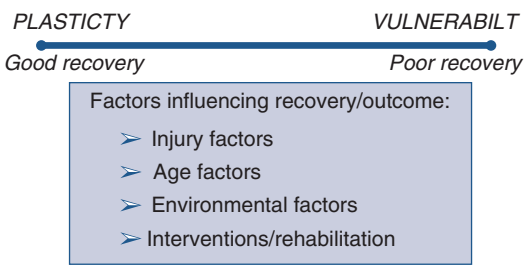
The early plasticity view contends that the brains of infants and young children are highly plastic, or flexible, relative to the brains of older children and adults, and as a result they are better able to overcome the adverse effects of brain damage. This perspective is supported by evidence from the most-studied types of brain damage, those associated with insults to areas of the brain associated with language. Since the 19th century, numerous reports have indicated that children who experience brain damage to the language areas of their left hemispheres before they are able to speak are eventually able to attain more advanced levels of language than are older children or adults who experience similar brain damage (Annett, 1973; Woods & Carey, 1979). Likewise, left-hemisphere brain injury for children who can already talk can produce an initial loss of language ability, but in many cases, language is recovered and children talk again at normal or near-normal levels. Studies conducted on adults have not found the same degree of recovery (Witelson, 1985). Even in young children, however, full recovery of language is rare, showing that the human brain is not completely plastic, even early in life (Witelson, 1987). Yet the evidence clearly shows that "there is a remarkable functional plasticity for language functions following brain damage in childhood in that the eventual cognitive level reached is often far beyond that observed in cases of adult brain damage, even those having extensive remedial education. These results attest to the operation of marked neural plasticity at least in the immature brain" (Witelson, 1987, p. 676). This pattern is also seen for milder forms of brain injuries, such as concussions. Children tend to recover from the effects of concussion (for example, headaches, memory loss) faster than adolescents and adults do (Yeats & Taylor, 2005). These

patterns of results are consistent with the early plasticity explanation of brain development and the view that if one must have brain damage, have it early, for a young brain is more likely than an older brain to recover normal function. This is the so-called *Kennard effect*, and it is based on the observations of Margaret Kennard, a pioneer in early studies on recovery of function from brain damage (Kolb, 1989).

Other research, however, has shown that the Kennard effect does not hold for all types of brain injuries, with some types of brain damage producing more long-lasting and negative consequences when experienced earlier rather than later in life, supporting the early vulnerability hypothesis. From this perspective, because the brains of infants and young children are becoming increasingly specialized with experience, early damage can alter the typical course of development resulting in serious disruptions of normal neural organization and functioning. Consider the analogy with the effects of teratogens (agents than can adversely affect the development of a fetus) on physical development. Exposure to a drug such as thalidomide interferes with the formation of limbs early in prenatal life, before arms and legs have been developed. The drug has no influence on limb development when exposure occurs later in prenatal life, after the limbs have been formed (K. L. Moore & Persaud, 2003). In fact, brain damage that occurs during the prenatal or early postnatal period typically results in permanent neurological impairment (V. Anderson et al., 2004; Riva & Cazzaniga, 1986), consistent with the early vulnerability perspective.

The early plasticity and early vulnerability views represent extremes of a continuum (see Figure 2.13), and research and theory notes that recovery depends on a wider series of variables than previously considered (V. Anderson et al., 2011; Kolb, 1989). For example, when the focus of damage is to an area of the brain involved with more general cognitive functioning such as attention, executive function, or intelligence as measured by IQ rather than with a specific

FIGURE 2.13 ■ The Human Brain Shows Both Early Plasticity and Early Vulnerability With Respect to Recovery of Function From Brain Damage. The Age at Which an Individual Experiences a Brain Insult Interacts With a Host of Other Factors to Determine the Likelihood of Recovery.



Source: Anderson, V., Spencer-Smith, M., & Wood, A. (2011). Do children really recover better? Neurobehavioural plasticity after early brain insult. *Brain, 134*, 2197–2221.

cognitive ability such as language, recovery is often greater the later the damage occurs (V. Anderson et al., 2009; Witelson, 1987). Evidence reviewed by Bryan Kolb and Ian Whishaw (1990) from both animal and human research demonstrates that younger children and animals show more permanent deficits than older children and animals after brain damage to the frontal lobes, which are associated with general processes such as those involved in IQ and executive function. For example, in one study, brain damage before the age of 1 year resulted in lower IQs for children than did similar brain damage that occurred after a child's first birthday (Riva & Cazzaniga, 1986). Another study reported greater reductions in IQ for children who suffered brain damage before the age of 5 than for those who suffered similar injury after age 5 (Kornhuber et al., 1985). In research with rats, Kolb and Whishaw (1981) reported that brain lesions inflicted shortly after birth resulted in a smaller adult brain (approximately 25% smaller) than did lesions inflicted on adult animals (approximately 12% smaller). Also, although infants and young children may demonstrate rapid recovery of cognitive function shortly after brain damage, deficits may appear years later. For example, toddlers with severe brain insults often display substantial recovery of "normal" abilities; however, these same children may show signs of cognitive deficits in their teen years, as day-to-day tasks become more demanding (M. Dennis, 1989).

Slow Growth and Plasticity

As I mentioned in Chapter 1, through most of the 20th century it was believed that if children suffered severe deprivation for much more than their first year after birth, they were destined to a life of intellectual impairment and psychopathology. Subsequent research with both human and nonhuman-animal participants has clearly shown that this is not true (Beckett et al., 2006; Suomi & Harlow, 1972). When the course of a young child's or young animal's life changes drastically, patterns of development can also be radically altered.

Let me provide one research example for the reversibility of the effects of negative early experience here. I presented some research on plasticity in Chapter 1, and the topic is discussed in greater detail with respect to intelligence in Chapter 13. With the political turmoil in Southeast Asia during the 1970s, many abandoned and sickly children from that part of the world were subsequently adopted by American families. Generally, follow-up interviews of adopted Asian children who were malnourished and socially deprived as infants revealed that their intellectual and social development was either at or above normal by early childhood (E. A. Clark & Hanisee, 1982; Winick et al., 1975). In the 1982 study by Audrey Clark and Jeanette Hanisee, for example, 25 adopted Asian children were given a test of verbal intelligence, the Peabody Picture Vocabulary Test (PPVT), and a test of social competence, the Vineland Social Maturity Scale (VSMS). The average age of the children at the time of testing was 44 months, and all the children had been in their adoptive homes for at least 23 months before testing. Before being adopted, most of the children had experienced physical and psychological deprivation. Sixteen of them were reported to have been malnourished sometime during infancy, with many displaying dehydration and muscle weakness. Despite their inauspicious beginnings, the children fared exceptionally well on the tests of verbal and social competencies. The national average on both the PPVT and the VSMS is 100. The adopted children's average scores were

120 on the PPVT and 137 on the VSMS. These children, impoverished and malnourished as infants, showed no residual signs of their early deprivation within 2 years of having been placed in upper-middle-class homes.

This plasticity of behavior and intelligence is attributed, in part, to the slow growth of the brain. As I noted earlier in this chapter, although the human brain is large relative to the rest of the body at birth, it continues to grow well into early adulthood. This prolonged immaturity provides humans the time necessary to master the complexities of social life. But it also provides the opportunity to change behavior and to acquire novel patterns later in life. Seen in this light, the extended immaturity of the human nervous system provides opportunities for resilience, behavioral flexibility, and plasticity unsurpassed by any other species (see Bjorklund & Pellegrini, 2002).

An immature brain means a slow and inefficient brain. Partly because of the extent of myelination and partly because of a paucity of experience, young children process information more slowly than older children do (see Kail, 1991). This slower speed of processing translates directly into less-efficient processing (Case, 1985) and means that more of younger children's processing is effortful in nature, in that it uses substantial portions of their limited mental resources (Hasher & Zacks, 1979; see Chapter 6). In contrast, more of older children's and adults' cognitive processing is automatic, in that it can be done quickly, without conscious awareness, and requires little or none of one's limited mental capacity. In other words, young children must work harder mentally to obtain the same results that older children can achieve more easily.

This inefficiency has its drawbacks, of course. You can't teach much of a complex nature to young children, you can't expect them to gain as much from experiences as older children do, and you can't rely on them to make many important decisions on their own. Despite the obvious disadvantages of a slow and inefficient brain, it also has its benefits. According to Bjorklund and Green (1992),

> Because little in the way of cognitive processing can be automatized early, presumably because of children's incomplete myelination, they are better prepared to adapt, cognitively, to later environments. If experiences early in life yielded automization, the child would lose the flexibility necessary for adult life. Processes automatized in response to the demands of early childhood may be useless and likely detrimental for coping with the very different cognitive demands faced by adults. Cognitive flexibility in the species is maintained by an immature nervous system that gradually permits the automization of more mental operations, increasing the likelihood that lessons learned as a young child will not interfere with the qualitatively different tasks required of the adult. (pp. 49–50)

This should not be seen as implying that the effects of early social or physical deprivation can always be reversed. As you'll see in later chapters, early experiences (or lack of them) can have relatively permanent, negative consequences. The experiences of infancy and toddlerhood establish patterns of behavior that can potentially influence the accomplishments of later years,

particularly when those experiences are stable over childhood. But the inefficiency of the young brain does offer children some protection from the perils of an early damaging environment.

Plasticity and Epigenetics

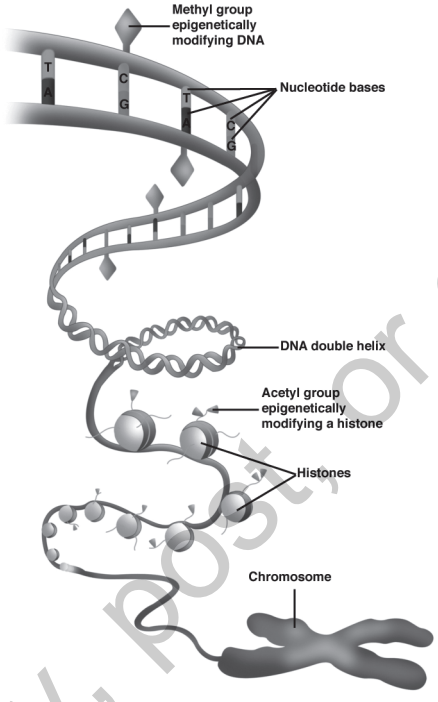
It should not be surprising that experiences alter the brain or that changes to the brain (and thus to cognition and behavior) differ with the nature of the experience and the age of the individual. What is perhaps more surprising is research showing how experience alters DNA expression and in turn behavior or cognition. The emerging field of **epigenetics** is helping to explain how, at the molecular level, experience affects behavior, and thus to develop a better understanding of plasticity.

At its simplest, epigenetics refers to how genes are expressed in different contexts (D. S. Moore, 2015). Epigenetics literally means "above genetics," and it has long been known that animals, including humans, inherit not only DNA from their parents but also chemical markers within the cytoplasm of cells that influence how genes are expressed. It was once believed that epigenetic effects were limited to the early stages of development, instructing genes to create specific structures and organs (ears and noses; hearts and spleens, for example), and then turning off those genes once the structures and organs had been formed. (Once you have two ears, you have no need for others.) Recent research has shown, however, that epigenetic processes occur throughout life, and they seem to be the mechanism responsible for modifying genetic activity as a result of experience.

There is a variety of biochemical processes involved in epigenetics, with **DNA methylation** being the most studied and best understood. Chemicals in the cytoplasm of cells from the methyl group can become attached to some of the nucleic acids (the chemical components of DNA), which affect the activity of that stretch of DNA, for example, causing a gene to produce more or less of a protein. Genes that are highly methylated produce less (or none) of the protein. Other chemicals in the cytoplasm can become attached to stretches of DNA, activating genes, a process called *acetylation* (see Figure 2.14).

Recent research has shown that methylation, or other epigenetic processes, is the primary mechanism by which experience modifies genetic activity and thus behavior. DNA methylation can be measured through blood or even scrapings from the inside of a person's cheek. Methylation of *promoter regions* of DNA (areas that act as on-off, or dimmer, switches for a gene) can be measured for a portion of the genome and related to experience and behavior. For example, in one study DNA methylation at age 19 for a subset of methylated regions was positively associated with health for adolescents with supportive parenting and higher socioeconomic status (Beach et al., 2016). Alternatively, DNA methylation for a specific gene known to be associated with particular psychological processes can be measured. For example, the *glucocorticoid receptor gene* (NR3C1) is associated with the regulation of the stress hormone cortisol. Several studies have demonstrated a relation between early stress, methylation of the glucocorticoid receptor gene, and subsequent development, including internalizing behavior (Kertes et al., 2016; Parades et al., 2016). For example, in a study by Sarah Romens and her colleagues (2015), 11- to 14-year-olds who had been maltreated as children displayed greater methylation

FIGURE 2.14 ■ A Schematic Diagram of DNA, Showing Nucleic Bases, the Double Helix, and Methyl Group and Acetyl Group Epigenetic Modifications.



Source: Moore, D. S. (2015). *The developing genome: An introduction of behavioral epigenetics*. Oxford University Press (p. 40).

to portions of the NR3C1 gene and with a gene associated with nerve growth factor than children who had not experienced maltreatment. Other research has reported relations between pregnant women's ratings of hardship during the 1998 Quebec ice storm and DNA methylation in their children's genes associated with the immune system 13 years later (Cao-Lei et al., 2014). Findings such as these are consistent with the argument made by developmental psychologist Marinus van IJzendoorn and his colleagues (2011) that child development can be defined as "experiences being sculpted in the organism's DNA through methylation" (p. 305). Behavioral epigenetics is in its infancy but holds great promise for understanding how experience changes the behavior of an organism and of the very process of plasticity itself.

DEVELOPMENTAL BIOLOGY AND COGNITIVE DEVELOPMENT

As the field of cognitive psychology evolved into cognitive science, people interested in mental functioning became increasingly aware of the need to coordinate the psychological level of explanation with the biological level. A similar realization has occurred in the field of cognitive development. For example, more than two decades ago, James Byrnes and Nathan Fox (1998) reviewed some of the new neuroscience techniques and research findings from the developmental neurosciences and concluded that we are on the threshold of a new revolution, equivalent to the cognitive revolution. Since then, the discipline of *developmental cognitive neuroscience* has emerged, which focuses on the study of typical cognitive and neurological development (M. H. Johnson & de Haan, 2011; Marshall, 2015). Perhaps its greatest contribution is elucidating the neurological mechanisms that underlie behavioral observations made earlier by psychologists. Admittedly, the presentation in this chapter is cursory, but I believe that it provides a foundation for the proper understanding of the ontogeny of human thought. Throughout the remainder of this book, I include contemporary neuroscience research in discussing a variety of aspects of cognitive development, from social cognition to executive function and memory, and I hope that this chapter will have served as an adequate introduction for placing this new brain-based research into perspective.

By acknowledging the importance of biological factors to cognitive development, I do not mean to suggest that the future of the field lies in biology. But having an idea of both the neural and evolutionary causes of behavior and development will help the psychologist ask better research questions and achieve a better understanding of development. For example, knowledge of the developmental relationship between brain and behavior has important implications not only for theories of cognitive development but also for societal practices. How pliable is human intelligence? When, in development, can children most benefit from certain educational experiences? Is earlier always better, or are certain sensitive periods for different aspects of perception and cognition distributed throughout development?

The study of cognition, including its development, has gone through substantial changes since its beginnings in the 1950s. I cannot be certain what the future holds, but it seems certain that part of the new paradigm will pay closer attention to the biological bases of cognition and cognitive development, including investigating brain development in a wider range of cultures and ethnic groups (Qu et al., 2021).

SECTION REVIEW

New *neuroimaging techniques*, such as high-density event-related potentials, PET, SPECT, fMRI, and fNIRS, are providing new knowledge about brain functioning and development.

Neuronal Development

- The nervous system consists of *neurons*, which transport chemical and electrical signals. Neurons consist of a cell body; *axons*, long fibers that carry messages away from the cell body to other cells; and *dendrites*, more numerous fibers that receive messages from other cells and transfer them to the cell body.

- Electrical messages are transmitted through *synapses*, facilitated by various *neurotransmitters*.
- Neurons go through at least three stages of development: *proliferation* (*neurogenesis*), *migration*, and *differentiation*.
- Synapse formation (*synaptogenesis*) is rapid during prenatal development and during the early months of postnatal life.
- A complementary process to synaptogenesis, *selective cell death*, or *apoptosis*, also occurs, with many neurons dying.
- *Myelin* is a fatty substance that surrounds axons, promoting faster transmission of electrical signals. Different areas of the brain begin and end the process of *myelination* at different times, and degree of myelination is related to certain sensory, motor, and intellectual levels of development.

How Do Young Brains Get Hooked Up?

- Some neural connections are made by all members of a species given typical experiences (*experience-expectant processes*), whereas other connections are made because of the unique experiences of an individual (*experience-dependent processes*).
- Neurons live (and form synapses with other neurons) or die as a function of use.
- Evidence suggests that areas of infants' brains are only weakly specialized for processing certain information (for example, language) but become more domain-specific in nature as a result of experience.

Development of the Neocortex and Plasticity

- The *neocortex* (or *cerebral cortex*) is divided into two hemispheres that are connected by the *corpus callosum*.
- Neuronal *plasticity* has been most clearly demonstrated in studies with animals, including those reared in deprived or enriched environments.
- With age, the plasticity needed to form new synapses declines, but it does not disappear. Humans possess *neural neoteny,* the expression of plasticity at the neuronal level into adulthood.
- Examination of the recovery of function after brain damage shows that the early plasticity and early vulnerability explanations represent ends of a continuum and that the degree of recovery of function from brain damage depends not only on the age at which the insult occurs but on a host of other interacting factors, including the area of the brain and cognitive abilities affected, the extent of the injury, and the experiences (including rehabilitation) of the individual following brain damage.
- Humans' prolonged immaturity contributes to our behavioral plasticity and to children's abilities to overcome the effects of deleterious early environments.
- New discoveries in the area of *epigenetics* reveals how experience affects behavior at the molecular level, helping to develop a better understanding of plasticity.

Ask Yourself . . .

1. Describe the three stages of neuronal development.
2. How are *synaptogenesis* and *apoptosis* complementary processes? What is their developmental function?
3. According to Greenough and colleagues, what are two ways in which experience influences neuronal development?
4. How does the development of the prefrontal cortex from birth to 2 years influence cognition and behavior?
5. What are some of the costs and benefits of early neuronal plasticity?

KEY TERMS

axon 54
behavioral genetics 47
biologically primary abilities 38
biologically secondary abilities 38
cerebral cortex 61
corpus callosum 61
critical period 44
deferred adaptations 33
dendrites 54
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